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DISEASES OF THE HEART

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BY

E. H. COLBECK, M.D. F.R.C.P. (LOND.), D.P.H. (CANTAB.),

AND

ARNOLD CHAPLIN, M.D. M.R.C.P. (LOND.)

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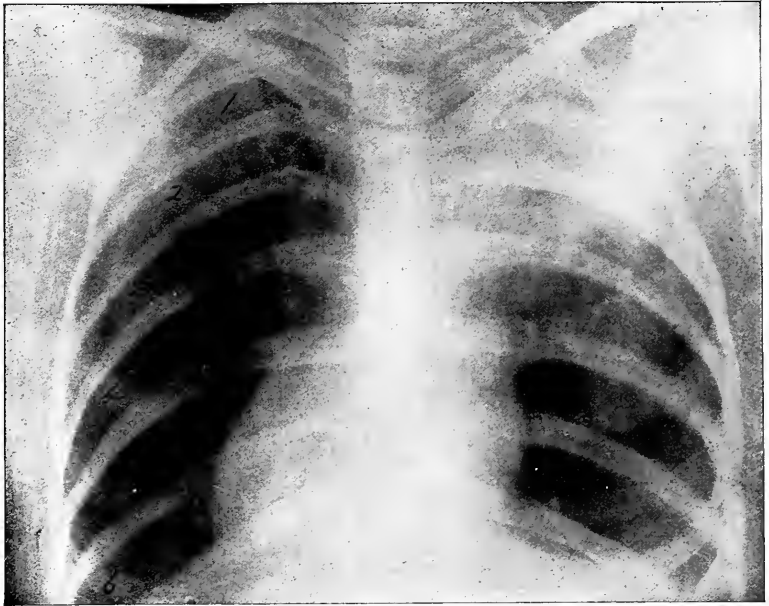
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SKIAGRAM OF THE CHEST SHOWING THE POSITION OF THE HEART
(Hugh Walsham)

The numbers 1-6 are placed on the posterior surface of the first six ribs in front.

DISEASES OF THE HEART

A CLINICAL TEXT-BOOK FOR THE USE OF
STUDENTS AND PRACTITIONERS OF MEDICINE

BY

EDMUND HENRY COLBECK

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ETC., ETC.

WITH FORTY-THREE ILLUSTRATIONS

SECOND EDITION. REVISED AND ENLARGED

W. T. KEENER & Co.,

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CHICAGO

1905

Lois Lyday

TO
SIR WILLIAM H. BROADBENT, BART.

M.D. (LOND.), F.R.S., F.R.C.P.

PHYSICIAN IN ORDINARY TO H.R.H. THE PRINCE OF WALES
CONSULTING PHYSICIAN TO ST. MARY'S HOSPITAL, AND THE LONDON FEVER HOSPITAL
LATE PRESIDENT OF CLINICAL, MEDICAL, NEUROLOGICAL, AND HARVEIAN SOCIETIES

WHOSE TEACHING HAS DONE SO MUCH TO

ADVANCE THE CLINICAL STUDY OF

DISEASES OF THE HEART

THIS BOOK

IS RESPECTFULLY AND GRATEFULLY

DEDICATED

PREFACE TO THE SECOND EDITION.

I HAVE taken advantage of the opportunity afforded by the demand for a Second Edition of "Diseases of the Heart" to make a few corrections in the text, and to add a little new matter, without in any way interfering with the general arrangement and character of the work.

A short account has been given of mechanical strain of the heart, and of the Stokes-Adams' syndrome; and the description of the treatment of chronic valvular disease has been somewhat amplified.

The instrumental determination of blood pressure which seems likely to occupy a prominent place in the clinical investigation of cardio-vascular disease has received but scant consideration; but in the present state of our knowledge it appeared hardly profitable to enter into a detailed description of this method of procedure.

The remaining additions that have been made do not call for comment, but it is hoped that they may enhance whatever usefulness the work may possess as a clinical guide to diseases of the heart.

E. H. C.

November 1904.

PREFACE

My interest in Diseases of the Heart dates from the time that I was appointed House Physician to Sir William Broadbent. I should have profited more by the teaching of this great master of clinical medicine had I possessed a better knowledge of the elementary and fundamental bedside features of cardio-vascular disease. It seemed to me at this time, and it does so still, that a book dealing with the clinical side of the subject of Heart Disease in a form suitable to the requirements of the student and newly qualified practitioner of medicine, would prove of very great service.

It has been my endeavour in the following pages to fulfil these conditions in as concise and systematic a manner as possible. In order to keep the size of the book within reasonable limits, compression of the subject-matter was unavoidable; but it is hoped that this process has not been carried so far as to interfere with the intelligibility of the text.

Controversial matter has to a large extent been avoided, and while this has entailed the exclusion of much new and interesting work, it has also permitted of much greater precision in the exposition of the subject, a feature of no small advantage to the student of clinical medicine.

I have freely consulted and made use of the writings of the leading authorities on Diseases of the Heart, and more

particularly those of Walshe, Peacock, Gairdner, Balfour, Broadbent, Sansom, Byrom Bramwell, Gibson, and Allbutt.

I am especially indebted to the masterly article on "Enlargement of the Heart" in *Keating's Encyclopædia of the Diseases of Children*, written by Dr. Mitchell Bruce, whose nomenclature with respect to the mode of production of dilatation of the heart I have adopted throughout the book.

The illustrations are for the most part original, but I have to acknowledge the kindness of Messrs. Charles Griffin and Co., and Messrs. Longmans, Green, and Co. in permitting me to make use of electrotypes of illustrations appearing in works published by them.

My thanks are due to Dr. J. G. Emmanuel, late Resident Medical Officer at the Chest Hospital, Victoria Park, for a large number of the pulse tracings.

Dr. Hugh Walsham has kindly placed at my disposal the skiagram of the chest, which appears as a frontispiece.

E. H. C.

LONDON, *January 11th*, 1901.



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DISEASES OF THE HEART

CHAPTER I

ANATOMY

Position and shape of Heart and Pericardium—Size and weight of the Heart—Dimensions of the Auricles and Ventricles—Relative position of the Cardiac Orifices and Valves—Relative size of the Orifices of the Heart—Relation of the Heart and its Orifices to the Sternum and Ribs

THE heart is situated in that portion of the cavity of the chest which is known as the middle mediastinum.

The Pericardium : its shape and connections.—The pericardium, wherein the heart is contained, is a cone-shaped membranous sac, which is attached by its base to the middle leaflet of the tendon of the diaphragm, and by its apex is continuous with the fibrous sheaths of the great vessels, and through them with the cervical fascia. This, the outer or fibrous layer of the pericardium, is lined by a serous coat, which is reflected along the great vessels on to the surface of the heart, and completely envelops the whole viscus. The space between the two layers of the serous covering is known as the cavity of the pericardium. Under normal conditions this space is potential rather than real, since the two layers of the sac are in close apposition.

The general shape of the heart is that of a blunt, flattened cone, the anterior convex surface of which faces upwards and forwards, while the more flattened posterior looks downwards and backwards.

The shape and position of the heart.—The base of the organ is directed upwards, backwards, and to the right, extending from the level of the sixth to the eighth dorsal vertebra, and the apex points downwards, forwards, and to the left. During life the apex beat is palpable in the space between the cartilages of the fifth

and sixth ribs, about $3\frac{1}{2}$ inches, or 9 cm., from the mid-sternal line. In the child the apex beat is situated in the fourth interspace.

Of the two borders of the heart, which are formed by the junction of the anterior and posterior surfaces, the one (*margo acutus*), longer and thinner, looks forwards and downwards, while the other (*margo obtusus*), shorter and more rounded, is directed backwards and to the left.

The axis of the heart is inclined to the horizon at an angle of 40° , and the greater portion of the organ is situated to the left of the median line.

The position of the heart is affected to some extent by posture, and by the degree of distension of the lungs. Except at its base, where it is attached to the great vascular trunks, the heart lies entirely free within the pericardial sac.

It will be observed that although the pericardium and heart are both conical in shape, the bases of the two cones do not coincide. Thus in the case of the pericardium the base of the cone looks downwards, while in that of the heart it is directed upwards, backwards, and to the right.

The relations of the heart.—The anterior surface of the heart is completely overlapped by the pleuræ and lungs, except over its lower part, where a roughly triangular area, to the left of the median line, remains uncovered. This uncovered portion of the wall of the heart, which corresponds with the area of superficial cardiac dullness, will be more accurately defined in a subsequent paragraph.

The size and weight of the organ.—The size and weight of the heart are liable to considerable variation, in accordance with the age, sex, stature, and muscular development of the subject, as well as with the method of removal that is adopted. The following figures, therefore, must be looked upon as representing average measurements only.

The weight of the adult male heart averages 11 ounces, or about 310 grammes; the weight of the adult female heart 9 ounces, or about 255 grammes. As a general rule, the adult has about 5 grammes of heart weight for each kilo of body weight; or, stated more exactly, the proportion of heart weight to body weight in the male is as 1 to 178, and in the female as 1 to 169 (Macalister).

External markings.—The external surface of the heart is divided into auricular and ventricular portions by a deep transverse groove—the auriculo-ventricular furrow. The division between the ventricles is shown by two shallow interventricular grooves, which, running from and at right angles to the auriculo-ventricular furrow, become continuous with one another a little to the right of the apex. It will thus be seen that the apex of the heart is formed entirely by the left ventricle. Moreover, owing to the position of the organ,

the right ventricle forms the greater portion of the anterior surface of the heart, and the left of the posterior.

Size and dimensions of the heart.—The capacity of each of the four cavities of which the heart is composed is approximately the same, and may be put at 100 cc., or about $3\frac{1}{2}$ ounces.

The walls of the different chambers vary greatly in thickness. The

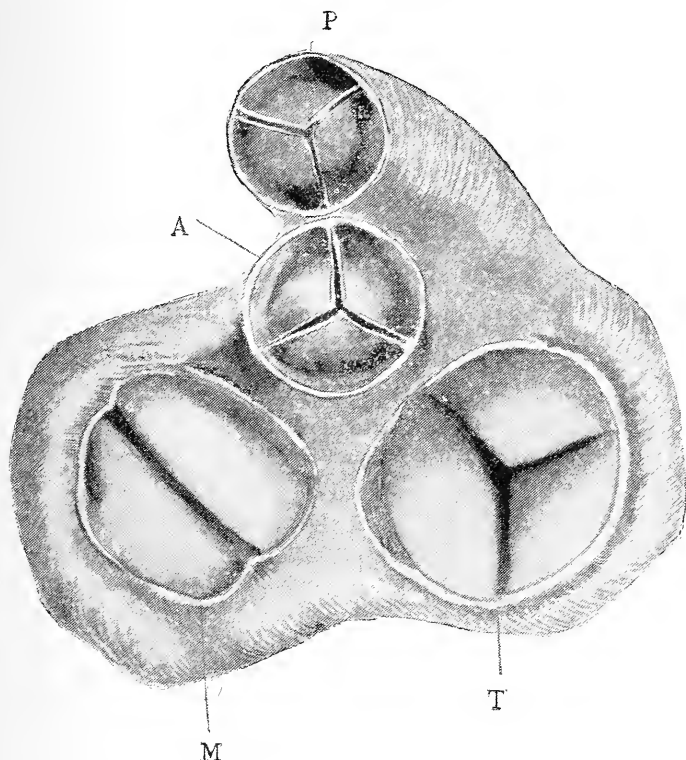


FIG. 1. SECTION THROUGH THE BASE OF THE HEART, SHOWING THE RELATIONS OF THE CARDIAC ORIFICES AND VALVES

P=pulmonary orifice ; A=aortic orifice ; M=mitral opening ; T=tricuspid opening

average thickness of the auricular walls is about 1 mm., or $\frac{1}{25}$ of an inch. The wall of the left auricle has a maximum thickness of 3 mm., or $\frac{1}{8}$ of an inch, and the wall of the right auricle 2.5 mm., or $\frac{1}{10}$ of an inch. The thickness of the wall of the left ventricle ranges between 4 mm. and 1 cm. ($\frac{1}{8}$ to $\frac{1}{2}$ inch), and of the right ventricle between 2 mm. and 4 mm. ($\frac{1}{12}$ to $\frac{1}{6}$ inch).

Relative position of the cardiac orifices and valves.—The relative position of the various orifices with their valves is most readily appreciated by means of a transverse section taken just above the ventricles, so that the structures exposed can be viewed from above.

The accompanying diagram illustrates such a section (Fig. 1).

Pulmonic orifice.—It will be observed that the conus arteriosus crosses the aortic opening obliquely from right to left, whence it happens that the pulmonic orifice is carried in front of and slightly to the left of the aortic.

Aortic orifice.—The orifice of the aorta lies in front of and between the two auriculo-ventricular openings.

The aortic and pulmonic orifices are each guarded by a valve consisting of three semilunar flaps, which are differently arranged at the two openings.

At the aortic orifice one flap is anterior and to the right, one anterior and to the left, and the other posterior and to the right; while at the pulmonic opening one is anterior and to the left, one posterior and to the left, and the third posterior and to the right.

Mitral opening.—The left auriculo-ventricular or mitral orifice is situated immediately behind and to the left of the aortic opening. It is guarded by a valve composed of a small posterior and a larger anterior curtain, the latter of which separates the mitral from the aortic orifice. The close proximity of the large anterior flap of the mitral valve to the orifice of the aorta is of some clinical importance, and will be again referred to.

Tricuspid opening.—The right auriculo-ventricular or tricuspid opening is closed by a valve consisting of three segments, of which one is placed anterior and to the left, a second to the right, and the third posteriorly.

It is worthy of notice that the dimensions of the aortic and pulmonic orifices are anatomically incapable of any alteration at the time of the closure of the semilunar valves. On the other hand, the size of the auriculo-ventricular openings is very much diminished during systole, in order to enable the valvular curtains to come into accurate apposition. This difference in the mechanism of closure of the two sets of openings has, as will be seen, important pathological and clinical bearings.

The following measurements (taken from Macalister's *Text Book of Human Anatomy*) are of some interest as showing the relative size of the orifices of the heart :—

		Males.		Females.
Area of the tricuspid orifice in sq. mm.	.	127·0	...	105·0
Area of the mitral orifice	„	96·0	...	86·8
Area of the aortic opening	„	70·6	...	65·2
Area of the pulmonic opening	„	68·5	...	64·5

Topography of the heart.—It has already been mentioned that nearly two-thirds of the bulk of the heart lie to the left of the median line of the body. The upward extent of the organ is represented on the surface of the chest by a horizontal line drawn slightly below the level of the second costal cartilages. The heart lies to the left of a vertical line drawn downwards from the sternal end

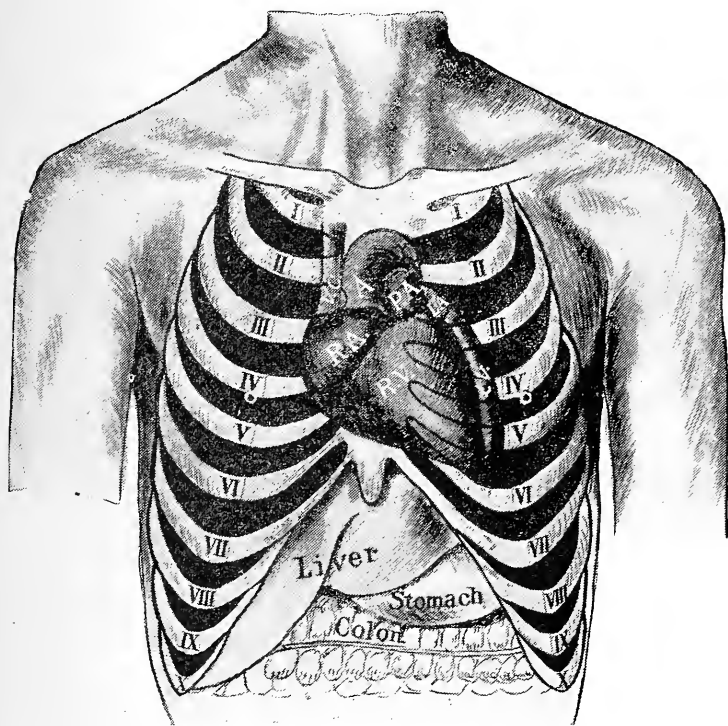


FIG. 2. RELATION OF THE HEART AND AORTA TO THE RIBS AND STERNUM

L.V. = left ventricle; R.V. = right ventricle; L.A. = left auricle; R.A. = right auricle;
A. = aorta; P.A. = pulmonary artery; V.C. = vena cava.

of the right clavicle, and to the right of an oblique line considerably convex upwards and outwards, drawn from the second costal cartilage about an inch from the left sternal border to the upper border of the sixth costal cartilage, 9 cm. or $3\frac{1}{2}$ inches from the mid-sternal line. It lies above a line, very slightly concave upwards, drawn from the upper border of the sixth rib, 9 cm. from the mid-sternal line, to the lower border of the cartilage of the right fifth rib, close to the sternal edge.

The width of the heart at the level of the fourth costal cartilage is about $4\frac{1}{2}$ inches, of which $1\frac{1}{2}$ inches lie to the right of the mid-sternal line, and 3 inches (nearly) to the left.

Area of deep cardiac dulness.—The whole of this area marks out what is known as the region of “deep cardiac dulness,” and corresponds approximately with the absolute size of the heart so far as its anterior aspect is concerned.

Area of superficial cardiac dulness.—By the term superficial cardiac dulness is meant the extent of the cardiac surface uncovered by the margins of the lungs, and in contact, through the pericardium, with the chest wall. This area is included in a triangle, which is bounded by the mid-sternal line, by a line drawn from a point on the mid-sternal line, opposite the level of the upper border of the fourth costal cartilages, to the lower border of the fifth left costal cartilage, at its junction with the rib, and by a line drawn horizontally inwards from this point to the mid-sternal line.

For practical purposes the area of superficial cardiac dulness may also be defined by a circle 2 inches in diameter, drawn from a point midway between the left nipple and the end of the gladiolus (Latham).

The relation of the different chambers and orifices of the heart to the area of deep cardiac dulness will now be described.

Right auricle.—The right auricle lies behind the sternal ends (about $1\frac{1}{2}$ inches from the mid-sternal line) of the third, fourth, and fifth costal cartilages, the second, third, and fourth intercostal spaces, and the right sternal edge. The apex of the right auricular appendix is situated in the middle line opposite the level of the upper border of the third costal cartilages.

Left auricle.—The only part of the left auricle that is visible from the front is its appendix, which is placed behind the second left interspace and upper part of the third costal cartilage about $1\frac{1}{2}$ inches from the left sternal edge.

Right ventricle.—The greater portion of the anterior surface of the heart consists of the wall of the right ventricle, which, measured along the left edge of the sternum, extends from just above the third rib to the lower border of the sixth costal cartilage. Its upper part is formed by the conus arteriosus, and is uncovered by lung.

Left ventricle.—The left ventricle forms the left border of the heart as high as the upper border of the third rib, and also the apex of the organ. The apex of the heart in adults is situated in the fifth intercostal space, 9 cm. or $3\frac{1}{2}$ inches from the mid-sternal line.

A diagrammatic representation of these relations is shown in the preceding figure.

Tricuspid opening.—The right auriculo-ventricular or tricuspid opening is behind the middle of the sternum opposite the fourth costal cartilages.

Mitral opening.—The left auriculo-ventricular or mitral orifice lies behind the sternum, to the left of the middle line, on a level with the upper border of the fourth costal cartilages.

Aortic orifice.—The aortic opening is situated behind the sternum to the left of the middle line on a level with the lower border of the third costal cartilages. The ascending portion of the aorta extends from this point to the level of the upper border of the second right costal cartilage at its junction with the sternum.

Pulmonic orifice.—The pulmonic orifice lies behind the upper border of the third left costal cartilage at its junction with the

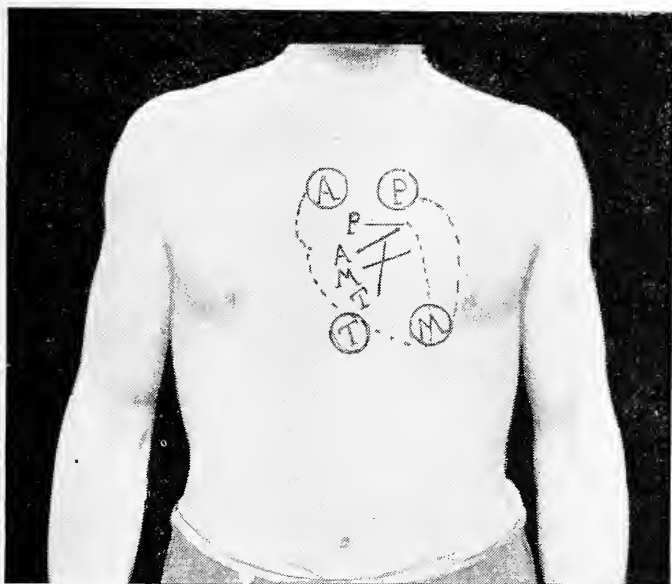


FIG. 3. THE POSITION OF THE VALVULAR ORIFICES, AND THEIR RELATION TO THE CARDIAC DULNESS

P=pulmonary orifice ; A=aortic orifice ; M=mitral orifice ; T=tricuspid orifice
 P (within circle)=pulmonary area ; A (within circle)=aortic area
 M (within circle)=mitral area ; T (within circle)=tricuspid area

sternum. The vessel ascends vertically from this point to the level of the second left costal cartilage, behind which it bifurcates.

The auriculo-ventricular sulcus.—The auriculo-ventricular sulcus is represented by a line drawn from the upper border of the

cartilage of the sixth rib on the right side, at its junction with the sternum, to the upper edge of the third left costal cartilage close to the sternal border.

The interventricular sulcus.—The anterior interventricular groove corresponds with an oblique line, slightly concave inwards, drawn from a point half an inch inside the apex, to the upper border of the third left costal cartilage, $1\frac{1}{2}$ inches from the left sternal edge.

It must be borne in mind that the foregoing landmarks are subject to considerable variation in different individuals, according to age, muscular and bony development, etc. In children the apex of the heart is situated a space higher than in adults, *i.e.* in the fourth intercostal space, and further to the left, so that it is often found in the nipple line, or even slightly outside this limit.

Aorta.—The first or ascending portion of the aortic arch extends from the lower border of the third left costal cartilage to the upper border of the second right costal cartilage at its junction with the sternum. The transverse portion of the arch runs across the sternum below the level of a line drawn horizontally through the middle of the manubrium, to the upper border of the fourth dorsal vertebra.

The third or descending portion of the arch lies to the left of the body of the fourth dorsal vertebra.

CHAPTER II

PHYSIOLOGY

Cardiac Cycle—Time relations and sequence of the various events which comprise Cardiac Cycle—Cardiac Impulse—Its position and cause—Sounds of the Heart—Their cause and duration—Nervous Supply of the Heart

THE term **cardiac cycle** is applied to that sequence of events which is included in each successive beat of the heart. It is composed of a contraction or systole of the auricles, a contraction or systole of the ventricles, and a pause or period during which all four chambers of the heart undergo relaxation, and then remain in a state of passivity (*i.e.* a condition in which the muscular walls neither contract nor relax) until the commencement of the succeeding cardiac cycle. This pause is called diastole.

The phases of contraction and relaxation on the right and left sides of the heart are, under normal conditions, exactly synchronous in the corresponding chambers.

The normal rate of the heart's action may be taken to be seventy-five beats per minute, so that each complete cardiac cycle is about eight-tenths ($\frac{8}{10}$) of a second in duration. The times occupied by the individual phases are *approximately* as follows:—

			Auricles.	Ventricles
Systole of the auricles	.	.	$\frac{1}{10}$ sec.	
Systole of the ventricles	.	.	—	$\frac{3}{10}$ sec.
Diastole of the auricles :				
Relaxation	.	$\frac{4}{10}$ sec.	} $\frac{7}{10}$ sec.	—
Passive interval	.	$\frac{3}{10}$ sec.		
Diastole of the ventricles :				
Relaxation	.	$\frac{1}{10}$ sec.	} —	$\frac{5}{10}$ sec.
Passive Interval	.	$\frac{4}{10}$ sec.		
Total Cardiac Cycle	.	.	$\frac{8}{10}$ sec.	$\frac{8}{10}$ sec.

It must be clearly understood that the *clinical* uses of the terms “systole” and “diastole” apply solely to these conditions in the ventricles. Consequently “systole,” as applied to the phases of the cardiac cycle, includes the ventricular contraction, and part of

the auricular relaxation ; while "diastole" comprises the ventricular relaxation, part of the auricular relaxation ; the passive interval, and the whole of the auricular contraction (see diagram Fig. 4).

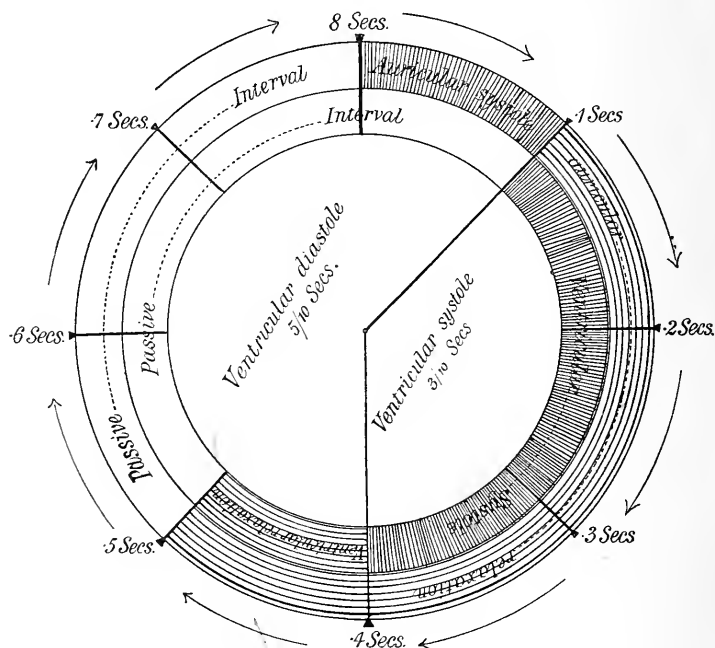


FIG. 4. DIAGRAMMATIC REPRESENTATION OF THE CARDIAC CYCLE

By the successive contractions of the auricles and ventricles the blood passes from the former into the latter, and from the latter into the arterial trunks. A reflux is prevented by the interposition of valves between the auricles and ventricles, and at the orifices of the aorta and pulmonary artery. The flaps of these valves come together and close the openings which they guard so soon as the blood pressure in front of them becomes greater than in the chamber immediately behind them. There is no valvular mechanism to prevent a reflux of blood from the auricles into the large veins during the auricular contraction, but immediately before and during the auricular systole there is a similar contraction of the muscular walls of those portions of the large veins which lie nearest the heart, and a consequent narrowing of their lumen, so that the blood takes the path of least resistance, and passes through the auriculo-ventricular openings into the ventricles.

Blood passes into the ventricles during the whole of the ventricular diastole largely in consequence of the positive pressure which prevails in the veins and auricles, but in some measure, no doubt, owing to the negative pressure which obtains in the ventricular chambers, by reason of the active dilatation of their walls. The final process of filling the ventricles is completed by the auricular systole.

The time relations and sequence of the various events which comprise the cardiac cycle are illustrated by the preceding diagram. The outer and inner circles represent the movements of the auricles and ventricles respectively. The radial and circular lines between the circles represent the contraction and relaxation of the auricles and ventricles respectively. The passive interval is left blank, while each eighth part of the circumference of the circles represents one-tenth of a second. The most constant feature of the cardiac cycle, with different rates of heart beat, is the duration of the ventricular systole. Hence it follows that an increase in the rate of the heart's action is obtained chiefly by means of the shortening of the diastole.

The cardiac impulse.—The cardiac impulse is the visible and palpable pulsation of the heart against the chest wall. It is synchronous with the systole of the ventricles, and can be most distinctly seen and felt in the fifth left intercostal space, about half to one inch on the sternal side of the vertical nipple line. The site and force of the cardiac impulse vary somewhat with the position of the body.

The cause of the cardiac impulse is briefly as follows. In the state of rest, during diastole, the heart lies with its axis directed obliquely downwards, so that the apex of the organ (in the upright position at all events) is in contact with the chest wall at the point mentioned above. During systole the heart, besides being tilted slightly upwards, moves forwards and to the right, whereby the apex is brought into closer contact with the thoracic parietes at a time when the wall of the ventricles suddenly becomes tense and hard. Furthermore, the antero-posterior diameter of the heart is increased during systole, and in this way the anterior surface of the ventricles is also brought nearer to the chest wall. The total effect of these changes is to bring the apex and adjacent portion of the cardiac wall, during systole, into somewhat violent contact with the thoracic parietes in the position of the apex beat.

The cardiac impulse, therefore, is the local displacement of the thoracic parietes produced by the more or less forcible impact of the apex and adjacent portion of the hardened and rigid cardiac wall during the ventricular systole.

A lengthening of the long axis of the heart is not concerned in the production of the apex beat, inasmuch as this diameter of the organ remains apparently unaltered during systole and diastole.

If the heart's impulse be allowed to impinge on the end of a lever, or on the membrane of a tambour in one of the many forms

of cardiograph, a tracing or impulse curve may be obtained, which will be considered more fully later (see p. 94).

The following is a normal cardiogram :—

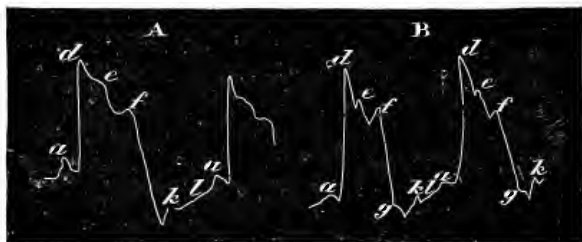


FIG. 5. NORMAL CARDIOGRAM
(Sansom after Galabin)

The sounds of the heart.—On listening over the region of the heart two sounds may be distinguished, which have been likened to the pronunciation of the syllables *lubb-dŭp*. The first sound, as indicated by the consonous syllable *lubb*, is comparatively dull, deliberate, and prolonged; while the second sound, represented by *dŭp*, is sudden, sharp, and short. The interval between the first and second sounds is very short, while that between the second and succeeding first sound is relatively of considerable duration. By direct observation it has been proved that the first sound is synchronous with the ventricular systole, and the second sound with the closure of the semilunar valves. The first sound of the heart is caused partly by the tension vibration of the segments of the auriculo-ventricular valves, consequent on the sudden closure of their orifices; partly by the so-called muscular sound, which is produced by the vibration of the muscular wall of the ventricles, consequent on their contraction; and partly by the vibration of the mass of blood in the ventricles and of the chordæ tendineæ.

The second sound is due partly to the tension vibration of the semilunar cusps, consequent on the simultaneous and sudden closure of the aortic and pulmonic valves, and partly to the vibration of the column of blood in the aorta and pulmonary artery.

The duration of the cardiac sounds, and more especially of the pauses between them, is subject to considerable variation, but the average times of these incidents, and their relation to the events comprising the cardiac cycle, are represented in the accompanying diagram. (Fig. 6.)

The nerves of the heart.—The nervous supply of the heart is derived from two sources, namely, from the pneumogastric or vagi, and sympathetic nerves.

The pneumogastric branches are as follow :—

1. The superior cardiac branches, two or three in number, leave the vagus between the superior and inferior laryngeal branches.
2. The inferior cardiac branches arise partly from the superior laryngeal nerve and partly from the main vagus trunk as it enters the thorax.

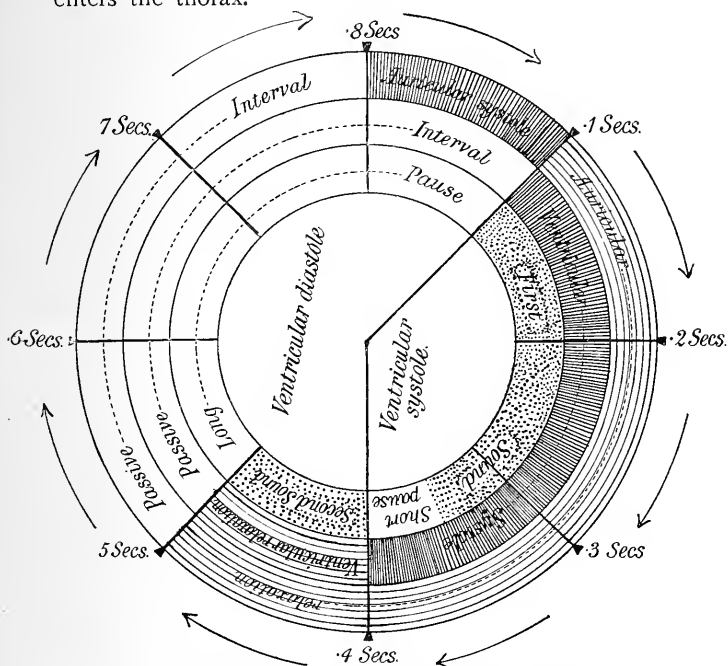


FIG. 6. DIAGRAMMATIC REPRESENTATION OF THE CARDIAC SOUNDS WITH REFERENCE TO THE CARDIAC CYCLE

The sympathetic branches are as follow :—

1. The superior cardiac branch from the superior cervical ganglion.
2. The middle cardiac branches (composed of small strands) from the middle cervical ganglion.
3. The inferior cardiac branches derived from several small strands from the inferior cervical and first dorsal ganglia.

Physiological experiment shows that in the dog, and presumably in man, the sympathetic fibres leave the spinal cord by the anterior roots of the second and third dorsal nerves, and then pass by the rami communicantes to the ganglia stellata (1st thoracic) and thence by the annulus of Vieussens to the inferior cervical ganglia, whence they are distributed to the heart *via* the cervical ganglia.

All three branches unite in the cardiac plexus, which may be described as consisting of a superficial and a deep portion. The former lies in front of the arch of the aorta on its concave border; the latter lies behind the aorta at a higher level than the superficial portion of the cardiac plexus.

From these two plexuses (*i.e.* superficial and deep) nerves pass directly to supply the auricular walls. The main distribution, however, is by two separate strands which accompany the right and left coronary arteries, and are called the right and left coronary plexuses respectively. The right coronary plexus supplies chiefly the posterior surface of the heart, while the left coronary plexus is distributed mainly over the left ventricle.

A large number of ganglion cells are interpolated along the course of the nerves composing these plexuses, more especially in the inter-ventricular and auriculo-ventricular grooves. From these ganglion cells a large number of fine nerve processes penetrate the substance of the heart to be distributed to the individual muscular fibres, as well as to the intermuscular, sub-endocardial, and sub-pericardial tissues.

The nuclei of the pneumogastric nerves, or rather those parts of them which supply the heart, *viz.* the accessory portions, are situated in the medulla oblongata, in close proximity to the respiratory and vaso-motor centres.

The sympathetic fibres have also a central connection with cells in the medulla and upper part of the spinal cord. The cells connected with the sympathetic fibres do not appear, however, to be aggregated into a definite centre, but seem rather to be distributed as a series of centres through the upper portion of the spinal cord, with, probably, a controlling centre in the medulla, situated near the pneumogastric nucleus.

The regulation of the beat of the heart, so far as this is determined by nervous influences, is effected for the most part, if not entirely, either directly or reflexly, through these centres. With regard to function the pneumogastric centres have been termed "cardio-inhibitory," and the sympathetic centres "cardio-accelerator" or "cardio-augmentor," inasmuch as those impulses which diminish the force and rate of the heart's action and prolong diastole, reach the heart by way of the pneumogastric nerves; while those impulses which increase the force and rate of the heart's action and shorten diastole reach the organ by way of the sympathetic fibres.

The action of the cardiac centres can be affected—

1. Directly, by the condition of the blood, by drugs, by alterations in blood pressure, etc.
2. Reflexly, by—
 - (a) Afferent impulses reaching the centres from the heart.

- (β) Afferent impulses from other organs, notably from the abdominal organs.
- (γ) Afferent impulses from the higher nervous centres originating in emotion, anxiety, grief, etc., or sensory impressions in the form of pain.
- (δ) Vaso-motor, respiratory, and other influences.

It must be borne in mind that, quite apart from nervous influences, the cardiac muscle has the power of independent rhythmical contraction; a power which is, however, but feebly developed in the mammalian heart. Nevertheless the beat of the heart can be, and is, profoundly modified by influences acting directly on the cardiac muscle, and possibly on the cardiac ganglia. The function of the cardiac ganglia is not fully known, but their influence on the regulation or modification of the heart's action is probably quite subsidiary.

The more important conditions which exert a direct influence on the cardiac muscle and thereby modify the beat of the heart are :—

1. Mechanical stimuli.—The heart is seldom exposed to mechanical stimuli; but the pressure that is not uncommonly exerted on the organ by a distended stomach, etc., may be classed under this head.
2. Alterations in the quantity and quality of the blood, both physiological and pathological; drugs, etc.
3. The degree of distension of the cardiac chambers, as, for instance, may be affected by respiration, alterations in blood pressure, or by disease in the form of valvular incompetence or stenosis of an orifice, etc.

Here the distension of one or other of the cardiac chambers increases the tension of its walls, and by this means gives rise, within certain limits, as in the case of skeletal muscle, to a more forcible contraction of the heart.

CHAPTER III

METHODS OF DIAGNOSIS

Enumeration of Methods—Section I. Symptomatology—Section II. *Ætiology*—Section III. The Physical Methods of Diagnosis—Sub-section I. Inspection ; general ; local—Sub-section II. Palpation ; *Præcordium* ; great vessels ; other organs—Sub-section III. Percussion ; Heart ; Pericardium ; great vessels ; other organs—Sub-section IV. Auscultation ; Heart Sounds and their Modifications ; Adventitious Sounds ; Vascular Sounds.

THE diagnosis in cases of heart disease is based on—

1. The symptoms.
2. The causal conditions indicated by the age, sex, occupation, and history of the patient.
3. The physical examination of the patient, which consists in the use of certain modes of procedure, distinguished under the titles of Inspection, Palpation, Percussion, and Auscultation. These physical methods of diagnosis should always be employed in the order in which they are named.

Each element in the diagnosis will now be considered under the headings of Symptomatology, *Ætiology*, and the Physical Methods of Diagnosis. A short account of the pulse and of the clinical uses of the sphygmograph and cardiograph will complete the chapter.

SECTION I

SYMPTOMATOLOGY

Disease of the heart may exist for a long time without giving rise to symptoms of any kind. Thus it not very uncommonly happens that the presence of a cardiac lesion remains unsuspected until some accidental circumstance, such as a medical examination for life insurance or for one of the public services, reveals its existence.

The symptoms of *morbus cordis* are sometimes referred mainly to the heart itself, but more often they are ascribed, for the most part,

to other organs, consequent on the disturbance of function that attends any mechanical derangement of their blood supply.

A systemic arrangement of the symptoms will be adopted in the following brief account of the subject.

Cardio-vascular system.—Apart from pericarditis and angina pectoris, præcordial pain is seldom a prominent feature of heart disease. A feeling of tightness, uneasiness, or pressure in the cardiac region, accompanied possibly by palpitation, or by the

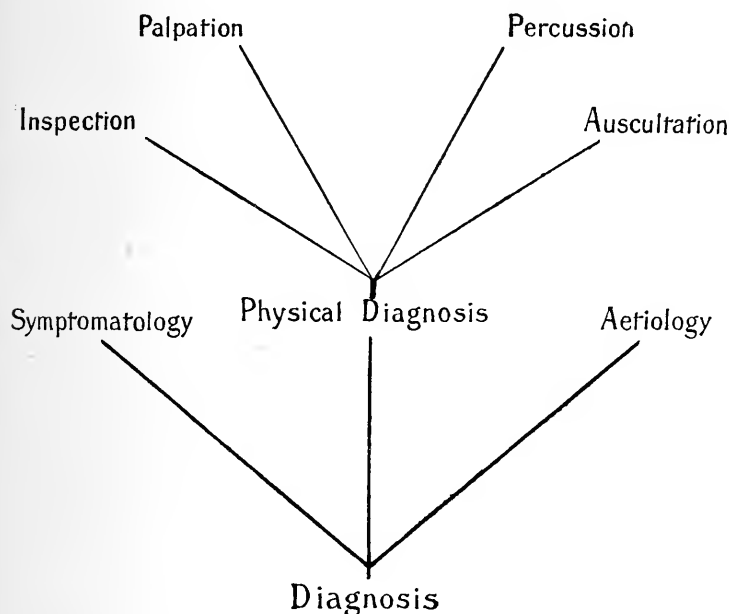


FIG. 7. DIAGRAMMATIC REPRESENTATION OF THE DIAGNOSIS

consciousness of an irregular or intermittent action of the heart, is sometimes complained of.

These symptoms are, however, more commonly due to nervous causes, or to digestive disorders, than to organic disease of the heart.

Pulsation may be felt in various situations, more particularly in the region of the head and neck, and is occasionally a source of much discomfort.

Noises in the ears are also experienced in connection with abnormal conditions of the circulation.

Hæmorrhage from the nose, lungs, stomach, uterus, etc., is by no means uncommon, and may lead to serious loss of blood.

Defective supply of blood to the extremities is the cause of the

cold hands and feet so commonly complained of by the subjects of heart disease. Cyanosis and dropsy will be considered under the heading of physical methods of diagnosis (*vide* Inspection).

Accidental symptoms, due to the embolic plugging of an artery in the brain, spleen, kidneys, or elsewhere, may occur at any period of the disease.

Respiratory system.—Shortness of breath on exertion is one of the earliest indications of commencing failure of the muscular power of the heart. The dyspnœa, which is at first excited and increased by effort, gradually becomes more pronounced as the cardiac weakness progresses, and in the final stages of the disorder may culminate in orthopnœa.

In some instances the patient suffers from paroxysmal attacks of difficulty in breathing, which are known under the name of cardiac asthma.

Rhythmic dyspnœa, in the form of Cheyne-Stokes' respiration, may precede the fatal event.

The pathology of cardiac dyspnœa will be discussed subsequently.

Cough, with a variable quantity and quality of expectoration, is often a troublesome symptom.

Hæmoptysis may occur more especially in mitral disease, and epistaxis is not uncommon in association with aortic insufficiency.

The subjects of heart disease are particularly liable to attacks of bronchitis, and in a less degree to pneumonia.

Digestive system.—It frequently happens that patients suffering from morbus cordis, notably in the form of mitral disease, seek medical advice in the first instance for the relief of dyspepsia.

The derangement of digestion usually takes the form of epigastric pain, or weight after food, nausea, vomiting, pyrosis, flatulence, and constipation, or irregular action of the bowels. Flatulent distension of the stomach reacts unfavourably on the heart by directly interfering with its free action, and in this way gives rise to palpitation and cardiac distress.

Congestion of the portal circulation is commonly associated with a greater or less degree of gastro-intestinal catarrh, which seriously aggravates the pre-existing dyspepsia.

Hæmatemesis sometimes occurs, and hæmorrhoids are frequently a cause of very great discomfort.

Pressure on the œsophagus, with difficulty in swallowing, may be due to a large pericardial effusion. Hoarseness or aphonia occasionally depends on a like cause.

Nervous system.—A feeling of depression and languor, disinclination for mental or bodily exertion, pain in the back and extremities, and weakness, twitching, and tremor of the muscles are among the early indications of a defective supply of blood to the central nervous system.

Headache, giddiness, noises in the ears, flashes of light before the eyes, and attacks of faintness or syncope are associated with disturbance of the cerebral circulation.

Sleeplessness is frequently a most distressing feature of diseases of the heart. On the other hand, drowsiness may be complained of, and sleep is often attended by unpleasant dreams. Delirium is occasionally observed, and is of serious import.

Emotional disturbances, mental changes, failure of memory, hallucinations, melancholia, dementia, etc., are not infrequently found in association with the different forms of valvular disease.

Hemiplegia, coma, and convulsions may be due to hæmorrhage in the brain from rupture of a blood vessel, to embolism of a cerebral artery, or to thrombosis in the cerebral veins.

The mode of onset of the symptoms considered in relation to the circulatory conditions at the time of their occurrence, the age of the patient, and the associated causal indications outside the nervous system, are the chief points in the differential diagnosis of these lesions.

Genito-urinary system.—The urine of heart disease (after failure of the organ) is scanty, high coloured, and throws down on cooling a copious deposit of urates.

It frequently contains albumen, hyaline or granular casts, bile, uric acid crystals, and occasionally blood. The characters of the urine of heart disease will be more fully considered under the heading of physical diagnosis (*vide* Inspection).

SECTION II

ÆTIOLOGY

The influence of the age, sex, occupation, and history of the patient on the incidence of heart disease will be considered under their respective headings.

Age.—Developmental malformations, and intra-uterine endocarditis account for the large majority of cardiac affections that are found during infancy.

In childhood, endocarditis, and pericarditis, of rheumatic origin, are comparatively common, and are apt to run a very insidious course. The rheumatic symptoms are usually slight, and unless great watchfulness is exercised may easily escape observation. The influence of chorea, a common disorder of childhood, in the production of endocarditis, is largely due to its rheumatic ancestry.

Certain of the acute specific fevers, notably scarlet fever, diphtheria, influenza, pyæmia, septicæmia, and measles are sometimes accompanied by inflammatory affections of the heart.

The endocardial inflammation in childhood usually attacks the mitral valve, rarely the aortic. During adolescence and early adult life (15–30) the acute inflammatory affections of the heart, due to

rheumatism, are more prevalent than at any other time. Furthermore, at this period the influence of anæmia in the production of cardiac disease is most marked, and at the same time functional disorders of the heart begin to make their appearance.

The mitral valve is more commonly affected than the aortic, but the relative immunity of the latter is not so great during this stage as in childhood.

Although the influences just mentioned are still operative, the middle period of adult life (30-45) introduces other important causes of cardiac disease, chief among which are physical overstrain, syphilis, and many other toxæmias, including the effects of excess in the use of alcohol and tobacco. Moreover, the effects on the heart and aorta of the high arterial tension that is associated with gout, contracted granular kidney, chronic lead poisoning, emphysema, etc., commonly begin to make their appearance during this and the succeeding decade of life.

In later adult life and during old age (45 and upwards) degenerative diseases of the heart and aorta become increasingly prevalent. True angina pectoris rarely occurs before the age of 40, and may therefore be fairly included among the cardiac lesions of the period under consideration.

Atheromatous affections of the endocardium may be primary, or they may be grafted on to pre-existing valvular disease.

The myocardium is frequently the seat of parenchymatous, fatty, or fibroid changes, as well as of other nutritional disturbances of a less defined nature, which are closely concerned in the production of failure of the heart, and dilatation of its cavities.

Sex.—Women suffer more frequently from chorea, anæmia, exophthalmic goitre, and functional affections of the nervous system than men, and are consequently more liable to the cardiac lesions associated with these morbid conditions. On the other hand, the influence of muscular overstrain, gout, syphilis, and alcoholism on the incidence of heart disease is more marked among males than females.

Occupation.—Persons who are exposed to cold and wet, as for instance washerwomen, out-door labourers, cabmen, etc., are more liable to suffer from the rheumatic manifestations of heart disease than other people.

All occupations that are attended by severe muscular exertion, and more especially those that entail sudden effort, are productive of enlargement of the heart, and, if the cause persist, of aortic and arterial disease, in the form of arterio-sclerosis and atheroma.

These conditions obtain principally among hammermen, miners, stokers, hill-climbers, puddlers, stonemasons, slaughterers, soldiers, and sailors, and to a less degree among athletes.

In the case of soldiers, and possibly in other instances, it is probable that a nervous element co-operates with the muscular overstrain in the production of the cardiac hypertrophy.

The enlargement of the heart, in the class of cases under con-

sideration, is due predominantly either to hypertrophy or to dilatation according as the state of cardiac nutrition is satisfactory or otherwise. Hypertrophy of the left ventricle is the condition usually found.

Aortic valvular disease, arterio-sclerosis, and atheroma are due to the strain to which the whole arterial system is exposed in cases of prolonged muscular exertion.

Persons of sedentary habits, who habitually eat and drink to excess, are frequently the subjects of atheromatous disease and of myocardial degeneration.

History.—A short account will be given under this heading of the various conditions of disease, and other morbid influences in the life-history of the patient that are causally related to affections of the heart.

Rheumatism, in one or other of its manifold phases, is by far the most important cause of heart disease. In many instances it is possible, indeed probable, that the cardiac affection is the sole manifestation of the rheumatic state. The endocardium, pericardium, and myocardium may be separately or simultaneously the seat of acute or chronic inflammatory changes in rheumatic cases. Endocarditis and pericarditis also arise in connection with certain of the specific fevers, notably with scarlet fever, less frequently with measles and erysipelas, and rarely with enteric fever and small-pox. Inflammation of the pericardium is a comparatively common complication of chronic Bright's disease, and it is occasionally observed in the course of pyæmia and pneumonia.

Malignant endocarditis, which in three-fourths of the cases is superimposed on previously existing valvular disease, occurs in association with pneumonia, puerperal fever, osteomyelitis, the specific fevers, phlebitis, and with other forms of septicæmia and pyæmia.

Pyæmia and phlebitis also account for a certain number of the cases of acute myocarditis.

The so-called "parenchymatous myocarditis" appears in the course of many of the acute febrile and infective processes, as for instance enteric fever, diphtheria, scarlatina, small-pox, typhus, relapsing fevers, septicæmia, and pyæmia.

Anæmia is a frequent cause of cardiac malnutrition and dilatation of the heart, which usually disappear with the restoration of a normal blood condition.

The comparatively common occurrence of mitral stenosis among women in the absence of a history of rheumatism has led to the belief that chlorosis may, in some instances, be a cause of this form of organic valvular disease.

Gout, chronic alcoholism, and syphilis are among the chief disposing causes of aortic valvular lesions, arteritis, and atheromatous affections of the arterial system. Degenerative changes in the myocardium, of a fatty and fibroid nature, are also frequently associated with alcoholism, syphilis, and other toxæmias.

Physical overstrain and protracted high arterial tension from any cause are productive of enlargement of the heart, and of arterial disease in the form of aortic valvular lesions, arterio-sclerosis, and atheromatous affections. Nervous disorders of the heart frequently owe their origin, in part at least, to physical overstrain.

Worry, anxiety, grief, and long-continued mental strain of any kind are sometimes the exciting cause of cardiac disease, and their occurrence adds greatly to the gravity of pre-existing affections of the heart.

The influence of heredity on the incidence of heart disease has also to be taken into consideration. Cardiac affections are hereditary chiefly in so far as their causes are hereditary. Thus rheumatism and gout, on which the large majority of the cases of heart disease depend, show a remarkable tendency to be transmitted from one generation to another. An hereditary tendency to high arterial tension and atheroma is also observed in some cases. The functional derangements of the heart which depend on neuroses of various kinds are likewise prone to run in families.

SECTION III

THE PHYSICAL METHODS OF DIAGNOSIS

So far as the examination of the heart is concerned, the physical methods of diagnosis range themselves under the four heads of Inspection, Palpation, Percussion, and Auscultation, which, as previously mentioned, should always be practised in this order. The data afforded by each of these methods will first be given in tabular form, and then discussed in detail.

SUB-SECTION I

INSPECTION

No complete or accurate physical examination of the heart is possible unless the entire chest is fully exposed.

The information afforded by inspection may be most conveniently classified under the two heads of

A. General B. Local

A. GENERAL.—This will be considered under the following scheme :—

- (a) *Appearance and attitude of the patient*
- (b) *Colour*
 - 1. Cyanosis or lividity
 - 2. Pallor
 - 3. Jaundice
- (c) *Dyspnœa*
- (d) *Dropsy*
- (e) *State of general nutrition*
- (f) *Urine*

B. LOCAL.—This will be discussed under the following arrangement :—

- (a) *Bulging or retraction of the præcordial region as a whole, or in part*
- (b) *Position of the apex beat*
- (c) *Visible pulsation*
 - 1. Cardiac
 - Præcordium as a whole, or in part
 - Epigastric
 - Displaced
 - 2. Vascular
 - Arterial
 - Venous
 - Capillary
 - Glandular

A. GENERAL

(a) Appearance and Attitude

The aspect of the patient suffering from morbus cordis is often very characteristic, and it should be noted in all cases. The facial appearance commonly associated with the more important cardiac lesions will be fully described under their respective headings. The attitude of the patient is frequently peculiar to his condition, and may have considerable diagnostic significance.

(b) Colour

1. Cyanosis or lividity.—This phenomenon, with clubbing of the fingers and toes, and coldness of the extremities, is usually associated with congenital malformations of the heart. It is, however, also observed, though usually to a less marked extent, in chronic disease affecting the mitral and tricuspid valves, and in lesions of the cardiac walls. Cyanosis, to any great degree, is rarely found independently of some form of heart disease. The discolouration of the skin in any given case is seldom constant, and may vary within very wide limits. It is increased by excitement, exertion, and lung complications, and diminishes under the influence of rest, sleep, etc.

Two theories have been advanced with the object of explaining the mode of production of the cyanosis. The one, proposed by Morgagni, attributes the blue tint to the general venous congestion caused by the obstruction to the free passage of blood through the right side of the heart; while the other, advanced by Hunter, ascribes it to the intermixture of venous and arterial blood. Both views have met with wide acceptance. There are, however, several very powerful arguments which favour the explanation afforded by Morgagni's theory. It has, in the first place, been clearly shown that no constant relation exists between the intensity of the cyanosis and the amount of intermixture of arterial and venous blood; more-

over, extreme cyanosis may exist without any possibility of intermixture. Furthermore it is noteworthy that in all cases of well marked cyanosis the conditions under which great venous obstruction may occur are present.

It would appear, therefore, that Morgagni's theory affords a fairly adequate explanation of the manner in which cyanosis is produced. At the same time it is probable that the intensity of the cutaneous discoloration is influenced by other factors, such as the amount of intermixture of arterial and venous blood; an insufficient aëration of blood by the lungs; dilatation of the cutaneous capillaries and an unusual transparency of the skin.

2. **Pallor.**—The frequent association of anæmia with morbus cordis is a matter of common observation, and its production, excluding accidental causes such as epistaxis, hæmoptysis, menorrhagia, etc., is accounted for in two ways, viz :—

1. That the chief causes of heart disease, as, for instance, rheumatism, the specific fevers, gout, etc., are *per se* fruitful sources of anæmia.
2. That in consequence of the particular lesion which exists, the circulation is inadequately maintained, and the proper assimilation of food and elimination of waste products is more or less defective, and hence the anæmia.

The most intense form of anæmia resulting from organic disease of the heart is that associated with aortic incompetence. Children suffering from morbus cordis are frequently the subjects of severe anæmia induced by repeated attacks of rheumatism.

The pallor affects the general surface of the skin, the conjunctivæ, and mucous membranes.

3. **Jaundice.**—This sign, which is seldom well-marked or constant, is usually observed after failure of the heart, and most commonly with mitral and tricuspid lesions. During the early stages of cardiac failure the jaundice is due to catarrh of the bile ducts, while at a later period of the disease it depends on organic changes in the liver, the result of long-continued venous congestion.

(c) **Dyspnœa**

Dyspnœa, by which is meant increased action of the respiratory muscles, both as regards frequency and degree of contraction, may be either inspiratory or expiratory, or a combination of the two conditions.

Speaking generally, cardiac dyspnœa is both inspiratory and expiratory.

With disease of the heart difficulty of breathing may be observed under several conditions :—

1. It may appear on exertion only, or as the result of some other external influence.

2. It may arise spontaneously, or without any obvious exciting cause.
3. It may be constant and postural, and it is then known as orthopnoea.
4. It may be paroxysmal, or rhythmic, in the form of—
 - (a) Cardiac asthma
 - (b) Cheyne-Stokes' respiration.

The value of dyspnoea as an indication of failure of the heart depends on the magnitude of the cause producing it. Dyspnoea which arises spontaneously, or as the result of very slight provocation, argues a greater degree of cardiac insufficiency than that produced by some definite and partially adequate cause. Orthopnoea, often associated with dropsy, usually indicates a considerable amount of dilatation of the heart from failure. The occurrence of Cheyne-Stokes' respiration with organic disease of the heart is usually but not invariably of fatal import.

The pathology of dyspnoea, though somewhat complicated, can be but briefly considered.

The movements of respiration are regulated by the respiratory centre, the activity of which depends on the quantity and quality of blood supplied to it. An excess of carbon dioxide, or a deficiency of oxygen in the blood, excites the centre to greater activity, with the result that increased action of the respiratory muscles is produced, or, in other words, dyspnoea.

Now, in disease at the mitral orifice there is always, owing to the position of the opening, more or less embarrassment of the pulmonary circulation, whereby the blood becomes insufficiently aerated, *i.e.* it contains more carbon dioxide and less oxygen than normal, and the consequent stimulation of the respiratory centre gives rise to dyspnoea.

This explanation, however, of the cause of cardiac dyspnoea is probably far from covering the whole of the ground.

A venous condition of the blood produces, also, through its effect on the vaso-motor centre, a general rise of systemic blood pressure by virtue of the contraction of the arterioles throughout the body. Von Basch has shown that increased systemic tension is invariably followed by a rise of blood pressure in the pulmonary circulation. He also points out that the accompanying distension of the pulmonary capillaries causes, so to speak, a general erection of the lung tissue, with enlargement of the lumina of the alveoli, and increased rigidity of their walls. As a consequence of these changes, increased inspiratory force is required to properly inflate the lungs, and expiration is difficult owing to the impairment of the elasticity of the alveolar walls, hence dyspnoea follows in proportion to the difficulties to be overcome.

It would therefore appear probable that in the production of cardiac dyspnoea, pulmonary engorgement acts in two ways:

(a) directly by a mechanical effect on the lungs, and (b) indirectly through the respiratory centre.

Dyspnœa produced by cardiac insufficiency assists the circulation inasmuch as each respiration pumps the blood from the venous system through the lungs (Leonard Hill).

The foregoing account, while offering a fairly adequate explanation of the causes of dyspnœa in disease at the mitral orifice, does not by any means exhaust the subject.

In pulmonic and tricuspid valvular lesions the mechanical interference with the circulation through the right heart and lungs will result in the insufficient aëration of the blood and the consequent stimulation of the respiratory centre with the production of dyspnœa.

In disease at the aortic orifice shortness of breath is always a prominent feature. Its production probably depends on several factors, chief among which are (1) the anæmia that is commonly associated with these lesions, and (2) the interference with the proper supply of blood to the heart and medulla, owing to the degenerative changes (arterio-sclerosis and atheroma) in the walls of the arteries supplying these structures, which are affected in common with the other arteries of the body in cases of aortic valvular disease.

In the production of cardiac dyspnœa an additional factor still remains to be mentioned, and that is the reflex disturbance of the nervous mechanism of respiration which must occur, to a greater or less degree, in all cases. Its influence is more obvious in the dyspnœa associated with disease of the aorta and its orifice and of the cardiac walls than of any other heart structure.

The precise manner in which this reflex effect is produced and the circumstances under which its action is brought into play have not yet received an adequate explanation, but there can be no doubt that sensory nerves supplying the heart, great vessels, and lungs are involved in the morbid processes affecting these structures.

A striking example of the reflex production of severe dyspnœa independently of pulmonary influences is seen in cases of acute inflammation of the aorta.

Orthopnœa is the condition in which the patient is obliged to adopt a semi-erect or sitting position, since any attempt to lie down is followed by severe and insupportable dyspnœa. The attitude of the patient is often peculiar to this form of laboured breathing, and is such that he can bring into action the accessory muscles of respiration to the greatest possible advantage. The upright position also facilitates the movements of the diaphragm, by reason of the descent of the liver, and other abdominal organs under the influence of gravity, and the same force may also retard the flow of blood in the inferior vena cava, and by this means relieve the engorgement of the right heart and pulmonary circulation.

The significance of orthopnœa has already been indicated.

The cause of cardiac asthma has not yet been fully determined, though doubtless in many instances it is due to toxic influences, the result of defective elimination by the kidneys.

The pathology of Cheyne-Stokes' respiration is still uncertain, though speaking generally the phenomenon is attributable to exhaustion of the respiratory centre, and the consequent deterioration of functional activity finds expression in a less specialized form of energetical manifestation, which, as in the case of all vital phenomena under similar conditions, tends to assume a rhythmical or periodic type.

(d) Dropsy

Dropsy of cardiac origin almost invariably begins in those portions of the body which are most distant from the heart. It commences, therefore, in the feet and legs and gradually spreads upwards. At an early period it invades parts like the scrotum and eyelids, where the subcutaneous tissues are looser, and the vessels less supported than elsewhere. Ultimately all the subcutaneous tissues may become infiltrated with fluid.

Effusion into the peritoneal and pleural cavities is not uncommon, and may occur at a comparatively early stage of the cardiac failure. The pericardium is affected less frequently, and usually at a later period of the disease.

Dropsy may be observed in almost any form of heart disease, but it is most commonly associated with mitral and tricuspid lesions, and with affections of the cardiac walls.

The pathology of cardiac dropsy is briefly as follows :—

In consequence of (a) the enfeeblement of the driving power of the ventricles and (b) the obstruction to the onward flow of blood through the right side of the heart, a condition of systemic venous engorgement is produced which leads to a general rise of intra-venous and capillary pressure. This state of the circulation favours an increased transudation of the fluid elements of the blood from the smaller vessels and capillaries into the connective tissue and lymph spaces, and a diminished re-absorption from them into the venous radicles. The process is in all probability intensified by changes in the composition of the blood, and by the consequent interference with the nutrition of the lining membrane of the capillaries and smaller vessels.

Under physiological conditions the lymph channels alone would suffice to carry off the increased quantity of fluid exuded from the capillaries, but the lymph circulation is also impeded, owing to (a) the general muscular enfeeblement, (b) the diminished aspirating power of the thorax, (c) the diminished suction power of the ventricles, and (d) the positive intra-venous pressure in the great trunks of the neck and thorax.

As the result, therefore, of the general venous engorgement, and the impeded lymphatic circulation, the fluid exuded by the capillaries and small vessels is unable to escape completely by either venous or lymphatic outlet, consequently it accumulates in the subcutaneous tissues and spaces, with the production of dropsy.

(e) State of General Nutrition

Lesions of the mitral valve are frequently attended by considerable emaciation, which depends, in a large measure, on the digestive derangements and the malassimilation of food associated with congestion of the portal circulation.

Aortic disease does not, as a rule, give rise to much disturbance of nutrition, though it is commonly found in persons of spare habit. Wasting, accompanied by anaemia, is sometimes a prominent feature of heart disease in children, and when extreme may, in conjunction with the respiratory difficulties, excite a suspicion of tuberculosis.

Lesions of the cardiac wall are in some instances found in association with general obesity.

(f) Urine

The urine in cases of morbus cordis presents no abnormal features until the heart fails. With the onset of cardiac failure it becomes scanty, high-coloured, and shows on cooling a copious precipitate of urates. Albumin is usually present in small quantity, and increases as the cardiac disability progresses. The specific gravity of the urine is raised, and the daily excretion of urea diminished. The occasional presence of blood in the urine is accounted for by the rupture of congested renal capillaries.

It is of great importance to distinguish the urine associated with failure of the heart from that due to acute or chronic inflammatory affections of the kidneys.

The latter are characterised by the presence of casts and other renal débris in the urine.

In acute inflammatory conditions of the kidney the urine is scanty, and contains a variable quantity of blood and large amounts of albumin and casts.

In chronic Bright's disease the urine is increased in amount, and contains little albumin and a variable, though usually small, number of casts.

The general manifestations of any morbid process which may injuriously affect the heart should be carefully noted during this part of the examination of the patient.

B. LOCAL

(a) Bulging or Retraction of the Præcordial Region as a Whole or in Part

Bulging of the præcordial area, which is not limited by the outline of the heart, may be due to rickets, spinal curvature, localized empyema, left pleural effusion, or to inflammatory conditions of the superficial tissues. The diagnosis of the cause of the

prominence in cases of this kind rests on the associated symptoms and physical signs, and as a rule presents no difficulties. Undue prominence of the præcordial region corresponding with the outline of the heart is most commonly caused by large pericardial effusions or by enlargement of the heart following valvular disease in early life. Bulging of the intercostal spaces in the cardiac area with widening of the interval between them is almost always the result of pericardial inflammation with effusion.

Forward displacement of the heart by means of a growth or other tumour situated behind the organ, and aneurism of the first portion of the aortic arch, are also occasional causes of præcordial bulging.

Flattening of the præcordial region not limited to the cardiac outline may be due to congenital malformation, to long-continued pressure, as in the case of shoemakers and joiners, or to retraction of the left lung.

Retraction of the præcordium caused by pericardial adhesions is usually most marked over the lower end of the sternum and adjacent portion of the left infra-mammary region. General præcordial retraction is sometimes due to universal pericardial adhesion, the result of pericarditis in childhood.

Bulging or retraction of the præcordial region, corresponding with different parts of the heart's area, is comparatively rare, and the cause of the condition may be deduced from the foregoing considerations.

(b) Position of the Apex Beat

The position of the apex beat has already been defined (see p. 1). The apex beat under normal conditions extends over an area included in a circle with a radius of about half an inch. It may be displaced from either (a) intrinsic or (b) extrinsic causes.

The further consideration of displacements of the apex beat will be found under the headings of "Palpation" and "Displacements of the Heart" (see pp. 34 and 35).

(c) Visible Pulsation

1. **Cardiac.** *Præcordium as a whole or in part.*—General præcordial pulsation of a diffused wavy character may often be observed in children with valvular disease, more especially when associated with pericardial adhesions. A similar phenomenon is occasionally seen in adults in cases of cardiac enlargement, and in those conditions in which the heart is brought into closer contact with the chest wall by means of the pressure of a growth or other tumour from behind, or as the result of the retraction of the lungs.

General systolic retraction of the præcordium can be due only to universal pericardial adhesion.

The most common cause of pulsation over a portion of the præcordial area is enlargement of the left ventricle, and the pulsa-

tion is then continuous and synchronous with the apex beat. Pulsation may occur, however, in this situation without any enlargement of the left ventricle, as in nervous excitation of the heart, Graves' disease, etc.

Enlargement of the right ventricle may give rise to pulsation between the left edge of the sternum and the apex beat.

Pulsation in the third left intercostal space close to the sternum may be due to enlargement of the left auricle, but it is probably more commonly the result of dilatation of the infundibulum of the right ventricle.

An enlarged right auricle may give rise to visible pulsation in the third and fourth interspaces to the right of the sternum.

Visible auricular pulsation may be either presystolic or systolic in time, but in the majority of instances it is systolic, owing to reflux from the ventricles.

Pulsation in the third, fourth, and fifth left intercostal spaces between the sternal edge and vertical nipple line is sometimes associated with anæmia, and may also depend on the uncovering of the heart by reason of the retraction of the left lung. Indeed, it may be stated generally that the presence or absence of præcordial pulsation largely depends on the extent to which the heart is covered by lung tissue.

Systolic retraction of the fourth and fifth intercostal spaces in the region of the apex beat is often associated with hypertrophied conditions of the left ventricle, and must not be regarded as a sign of pericardial adhesion.

Systolic retraction of the ribs, costal cartilages, or sternum is indicative of pericardial adhesion. Of much greater importance, however, in the diagnosis of this condition is diastolic retraction of the intercostal spaces, which can hardly be produced by anything but pericardial adhesion. Systolic and diastolic retraction of spaces may be associated.

In the diagnosis of pericardial adhesion the signs afforded by inspection and palpation should be considered together, inasmuch as, at first sight, the data obtained by the two methods appear to be contradictory, *i.e.* the visible recession of the chest wall is accompanied by a palpable impulse.

Epigastric pulsation.—This phenomenon can be produced by the forcible pulsation of a normal right ventricle, such as is occasioned by simply holding the breath, or by any temporary pulmonary obstruction.

Visible epigastric pulsation commonly means either enlargement (hypertrophy and dilatation) of the right ventricle, or displacement downwards of the heart, or, as in pulmonary emphysema, a combination of the two conditions.

Other accidental causes of epigastric pulsation are: (1) neurotic pulsation of the abdominal aorta; (2) aneurism of the upper part of the abdominal aorta, or a tumour in front of this portion of the

vessel; (3) pulsation of the liver; (4) systolic recession of the epigastrium due to pericardial adhesion.

The differential diagnosis of the cause of epigastric pulsation is not usually a matter of any great difficulty.

Immobility of the upper portion of the epigastric triangle is, according to Sir William Broadbent, always associated with extensive pericardial adhesion.

Displaced pulsation.—The cardiac impulse may be displaced upwards or downwards, to the right or left, conformably with alterations in the position of the heart. In extreme instances there is a complete absence of pulsation over the normal situation of the organ, which may be seen beating in some unusual position.

The causes of displacement of the heart will be considered under the heading of "Palpation."

2. Vascular pulsation. *Arterial pulsation.*—Pulsation above the level of the third rib, if systolic in time, is generally of arterial origin.

If in the second right intercostal space, close to the sternum, it is probably due to dilatation of the ascending portion of the aorta. If in the second left interspace, the cause is usually exposure of the pulmonary artery owing to retraction of the left lung.

Pulsation due to aneurism of the arch of the aorta may appear to the right or left of the sternum, or in the episternal notch and middle line, according to the part of the vessel that is affected. Pulsation in the episternal notch may be visible under perfectly normal conditions.

Aneurism of the innominate artery may give rise to pulsation in the neighbourhood of the sternal end of the right clavicle.

Pulsation due to aneurism of the subclavian arteries may appear above or below the clavicle. The presence of pulsation beneath the outer ends of either clavicle, often visible in thin subjects, is of no clinical importance.

Visible and forcible pulsation of the arteries at the root of the neck may occur under many conditions. It is commonly seen in nervous affections of the heart, in anæmia, and after great exertion or mental excitement.

Visible pulsation of the arteries generally is particularly characteristic of aortic insufficiency, and is then often associated with a locomotion forwards of the vessels. The latter phenomenon is well seen in the case of the temporal arteries which, in addition to the forward displacement, undergo a peculiar vermicular movement with each beat of the heart.

A similar condition, though very much less marked, may occasionally be seen in the arteries of old people in whom the arterial wall is rigid, and the contraction of the left ventricle sudden and badly sustained.

Venous pulsation.—Except in some cases of anæmia and chlorosis, and in rare instances in healthy people, pulsation in the veins of the neck is accompanied by venous distension. Apart from these exceptions venous pulsation is either auriculo-systolic or ventriculo-systolic, or both, and is indicative of obstruction at the tricuspid orifice, or of incompetence of its valve. The most common cause of venous pulsation in the neck is incompetence of the tricuspid valve, the result of failure of the right ventricle. Tricuspid incompetence may exist without venous pulsation, provided the amount of regurgitation is slight, and the venous valves remain competent; and, conversely, slight pulsation may occur in the veins of the neck without any tricuspid incompetence, the pulsation being the result of the auricular systole, or of the vibration of the column of blood in the veins resting on suddenly closed tricuspid valve-segments. It is important to distinguish venous pulsation in the neck from arterial pulsation in the same situation. If the finger placed lightly on the external jugular vein at the root of the neck obliterates the pulsation it is probably venous; if it continues it is probably arterial. Moreover, if the vein emptied by passing the finger lightly over the vessel from below upwards is seen to fill from below, the pulsation is certainly venous and obviously the result of regurgitation through the tricuspid orifice. Venous distension of the large trunks at the root of the neck may also be caused by chronic lung disease, by the pressure of a tumour on the great veins in the thorax, or by tricuspid stenosis. Diastolic collapse of the veins at the root of the neck may occur in cases of adherent pericardium. The phenomenon is supposed to depend on the formation of fibrous adhesions involving the great veins. Owing to the stretching of these adhesions during the systole of the ventricles the lumen of the vessels is narrowed, and thereby the onward flow of blood is hindered. During diastole, on the other hand, the fibrous bands being relaxed, there is a sudden rush of blood into the heart, consequent on the removal of the obstruction, and on the suction power of the ventricles. Hence the venous collapse.

Occasionally a pulse may be seen in the peripheral veins, such as those on the dorsum of the hand. The phenomenon is best seen in cases of aortic regurgitation, but may occur to a slighter extent in other conditions.

Capillary pulsation.—This is observed in cases of aortic incompetence, in which the amount of regurgitation is considerable and the power of the left ventricle is still good.

A portion of skin, preferably of the forehead, should be rubbed with the finger-nail, or end of the stethoscope, and the flush thus produced will be seen to pulsate synchronously with the heart's beat, becoming alternately red and pale. The same appearance may often be seen through a thin slip of glass gently pressed on the everted lip, or tip of a finger. Its production depends on the forcible injection of blood into the lax arterial system and the sudden backflow

into the ventricle, owing to the absence of support to the column of blood by reason of the incompetence of the aortic valve.

Glandular pulsation.—Occasionally, as in Graves' disease, slight pulsation may be observed in the thyroid gland.

In some cases of tricuspid regurgitation the whole liver may be seen to pulsate. This phenomenon is of somewhat rare occurrence, and is indicative of a considerable degree of tricuspid insufficiency. Care must be taken that transmitted pulsation from the right ventricle is not mistaken for true expansile pulsation of the organ. (See Palpation.)

SUB-SECTION II

PALPATION

For the purposes of palpation of the heart the patient may be in the erect or recumbent position, but it is advisable, when possible, to make use of both postures in the examination.

The præcordium should be palpated in the first instance by the whole hand placed flat upon the chest, and the signs thus perceived may then be localized by means of one or two fingers. The data afforded by inspection and palpation should finally be compared.

Palpation of the heart will be considered under the following scheme :—

A. PRÆCORDIUM

- (a) *The apex beat and impulse of the left ventricle*
- (b) *The impulse of the right ventricle*
- (c) *The auricular impulse*
- (d) *The diastolic impulse*
- (e) *Closure of the semilunar valves*
- (f) *Vibrations or thrills*
 - 1. Endocardial
 - Systolic
 - Diastolic
 - Presystolic
 - 2. Exocardial
 - Friction fremitus
- (g) *Fluctuation*

B. GREAT VESSELS

- (a) *Aneurismal pulsation*
- (b) *Diastolic back shock*
- (c) *Thrills*
 - 1. Arterial
 - 2. Venous
- (d) *Tracheal tugging*

C. OTHER ORGANS

A. PRÆCORDIUM

(a) The Apex Beat and Impulse of the Left Ventricle

The apex beat of the heart, which is the expression of the contraction of the left ventricle, should be investigated with respect to its site, extent, and character.

The site of the apex beat. The angulus Ludovici forms a useful guide to the level of the second rib, immediately below which is the second intercostal space, and from this the rest of the ribs and spaces can be easily counted.

In adults the apex beat is situated in the fifth intercostal space $3\frac{1}{2}$ inches from the mid-sternal line. Inability to palpate the apex beat in its normal situation may be due to (a) the covering of the heart by lung, as in emphysema; (b) unusual thickness of the chest wall, depending on subcutaneous fat, mammary tissue, etc.; (c) weakness of the left ventricle, and (d) displacement of the heart and apex beat.

The heart and apex beat may be displaced either from (a) intrinsic or (b) extrinsic causes.

The intrinsic causes of displacement of the apex beat are hypertrophy or dilatation of the ventricles, or, as more commonly happens, a combination of the two conditions.

The apparent upward displacement of the apex beat in pericardial effusion is, in all probability, due to the separation of the lower portion of the left ventricle from the chest wall by a layer of fluid. If the patient leans well forward, by which means the ventricle is brought nearer to the chest wall, the apex beat may become palpable in its normal situation. This manœuvre is frequently sufficient to render the apex beat easily palpable in other cases in which, under the ordinary conditions of examination, the sign cannot be perceived, or is very indistinct.

In hypertrophy of the left ventricle the apex beat is displaced downwards and slightly outwards; in dilatation it is displaced outwards and slightly downwards. As a rule the two causes of displacement are variously combined.

In some instances of well-compensated aortic stenosis there is reason to believe that the apex beat may not be palpable; but this point will receive further consideration under the account of the valvular disease in question.

Enlargement of the right ventricle displaces the apex beat outwards.

The extrinsic causes of displacement of the heart and apex beat may be congenital or acquired.

Congenital displacement of the heart will be considered under the head of "Malformations and Misplacements of the Heart."

The acquired causes of displacement of the heart and apex beat operate by means either of pressure or traction on the organ, which

may in consequence be dislocated towards the right or left, upwards or downwards, backwards or forwards.

Displacement of the heart and apex beat to the right.—This may be due to the pressure of fluid, gaseous, or solid accumulations in the left pleural cavity, or to extensive consolidation of the left lung.

The heart may be drawn towards the right by retraction of the right lung following pleural effusion; by the contraction of pleuro-pericardial adhesions, of pulmonary cirrhosis, or of cavities in phthisis; and by collapse of the right lung from pressure on the main bronchus on the right side.

Displacement of the heart and apex beat to the left.—Dislocation of the heart to the left may depend on fluid, gaseous, or solid accumulations in the right pleural cavity, or on extensive pneumonic consolidation of the right lung. It may also be due to contraction of the left lung from any of the causes already enumerated. Aneurism of the first or second portion of the arch of the aorta is an occasional cause of displacement of the apex beat to the left.

Displacement upwards of the heart and apex beat.—This form of displacement may be caused by fluid, gaseous, or solid collections in the abdominal cavity, such as ascites, tympanites, tumours of the liver, the pregnant uterus, and abdominal tumours of all kinds. Retraction of the upper portion of either lung may also give rise to upward displacement of the heart.

Displacement downwards of the heart and apex beat.—Hypertrophic emphysema is the commonest cause of this variety of cardiac displacement, which is very frequently observed. Tumours at the base of the heart and collapse of the abdominal viscera also lead to downward dislocation of the organ.

Displacement backwards of the heart and apex beat.—This variety of displacement, which is very rare, may be produced by large pericardial effusions, and by tumours or inflammatory accumulations in the anterior mediastinum.

Displacement forwards of the heart and apex beat.—Forward dislocation of the heart is very uncommon, but it is occasionally observed as the result of the pressure of a tumour situated in the posterior mediastinum.

It will thus appear that the determination of the site of the apex beat is a point of the greatest value in the diagnosis, not only of diseases of the heart, but also of the lungs, and it not infrequently happens that the careful location of this sign supplies the key to the elucidation of many obscure affections of the chest.

The extent of the apex beat.—The apex beat may be localized or diffuse. Speaking generally it may be stated that in hypertrophy of the left ventricle the apex beat is defined and localized, whereas in dilatation it is hesitant, uncertain, and diffuse. A combination of hypertrophy and dilatation often gives rise to a wide area of pulsa-

tion, which occasionally involves a large portion of the anterior surface of the left chest.

The character of the apex beat.—The apex beat may convey the sensation of a powerful prolonged heave or thrust, or of a short, sharp tap, or slap. The former phenomenon is indicative of hypertrophy, the latter of dilatation of the left ventricle. A typical example of the first condition is provided by a fully compensated case of aortic insufficiency, and of the second, by fatty degeneration of the heart wall. Various combinations of the two above-mentioned conditions may be observed, with corresponding modifications in the character of the apex beat.

The importance of a careful estimation of the character of the apex beat must be insisted on, since it is a factor of the first magnitude in the forming of a prognosis in valvular and myocardial diseases of the heart.

The effect of respiration and change of posture on the position of the apex beat should be carefully noted in all cases.

(b) The Impulse of the Right Ventricle

In cases of enlargement of the right ventricle the degree of vigour of the ventricular contraction may be gauged by palpation in the epigastric region. In disease at the mitral orifice, whether obstructive or regurgitant, the task of maintaining the pulmonary circulation, or in other words of providing compensation, falls on the right ventricle; hence the importance of investigating the strength of the ventricular impulse.

The character of the impulse would have to be considered in conjunction with the competency or incompetency of the tricuspid valve.

(c) The Auricular Impulse

Occasionally it is possible, by placing the tips of the fingers in the second, third, and fourth right intercostal spaces close to the sternum, to feel an impulse resulting from the contraction of the right auricle. The impulse is presystolic in time. A similar phenomenon is said to have been observed, in children only, in the corresponding situation on the left side, and has been ascribed to the contraction of the left auricle.

(d) The Diastolic Impulse

This is very rarely observed, but when present it is felt as an impulse during the diastole of the ventricles. It is usually found in association with a variable degree of ventricular dilatation, and with a sudden sharp systole of the heart. It is more commonly observed in children than in adults. A peculiar diastolic shock or retraction, palpable over the apex or right ventricle, may be due to the presence of pericardial adhesions.

(e) The Closure of the Semilunar Valves

The closure of the pulmonic semilunar valves may sometimes be felt in those conditions in which, owing to the retraction of the left lung, the pulmonary artery comes nearer to the chest wall. Under these circumstances the artery is often somewhat dilated. The closure of the aortic valve may also be felt in cases of high arterial tension with dilatation of the ascending portion of the aorta just above the sigmoid cusps.

(f) Vibrations or Thrills

1. **Endocardial.**—The peculiar and characteristic vibratory or quivering sensation, which may be felt by the hand placed over the heart, is known as a thrill.

In the examination of a thrill attention should be directed to (*a*) its position and (*b*) its time relation with respect to the cardiac cycle, which is determined by placing a finger on the apex beat, or carotid pulse, while the other hand palpates the thrill.

A thrill that is synchronous with the ventricular impulse is called systolic, while one that alternates with the impulse is termed diastolic. The diastolic thrill may be felt throughout the diastole of the ventricles, or it may be palpable during the early part only of the diastole, when it is called an early diastolic or post-systolic thrill, or during the late part only of the diastolic period, when it is known as a late diastolic or presystolic thrill.

A systolic thrill felt over the apex beat indicates mitral incompetence; a presystolic or diastolic thrill in this situation is indicative of mitral obstruction, but, as will be shown later, it may be associated with aortic regurgitation.

Thrills felt over the base of the heart; in the second right intercostal space close to the sternal edge, or in the episternal notch, are indicative of disease at the aortic orifice. If systolic, the opening is obstructed, if diastolic, the valves are incompetent. Diastolic thrills due to aortic insufficiency may also be felt along the right sternal edge as low as the fifth costal cartilage, and in the second, third, and fourth left intercostal spaces close to the sternum, as well as at the apex beat. Dilatation of the aorta just above the valve may give rise to a systolic thrill in the second right interspace.

Thrills felt in the second left intercostal space or over the third left costal cartilage close to the edge of the sternum, indicate disease at the pulmonic opening. If systolic, the orifice is obstructed, if diastolic, the valve is incompetent.

A presystolic thrill, palpable in the fourth and fifth intercostal spaces close to the sternum, or over the xiphoid cartilage, is indicative of tricuspid stenosis.

Congenital disease of the heart not infrequently gives rise to a præcordial thrill, which is more commonly felt over the base of the organ than at the apex.

The intensity of a thrill is often markedly affected by alterations in the position of the patient.

2. **Exocardial.** *Friction fremitus.*—This sign, which is the result of the rubbing together of roughened pericardial surfaces, is generally felt earliest over the base of the heart, or over the region of the fourth left costal cartilage. It is commonly systolic in time, but it may have a double rhythm, *i.e.* systolic and diastolic, and gives the sensation of a rubbing movement to the hand.

Pleuro-pericardial friction fremitus is sometimes felt in the region of the apex beat and along the left border of the heart as high as the third rib.

(g) Fluctuation

Fluctuation is occasionally felt over the præcordial region in cases of long-standing, large pericardial effusion.

B. GREAT VESSELS

(a) Aneurismal Pulsation

It has already been stated that pulsation above the level of the third rib is generally aneurismal. The features to be observed on palpation are the rhythm, which will be systolic, and the character of the pulsation, which will be expansile.

(b) Diastolic Back Shock

It has already been mentioned that a diastolic back shock may be felt over the aortic cartilage (second right) in cases of high arterial tension. A similar phenomenon may be observed in aneurismal conditions of the aorta.

(c) Thrills

1. **Arterial.**—A thrill, systolic or diastolic in time, may be felt over an aneurism.

2. **Venous.**—A venous thrill may often be felt in the jugular veins at the root of the neck in cases of chlorosis and anæmia. The proper appreciation of these thrills necessitates the gentlest palpation.

(d) Tracheal Tugging

This phenomenon, which is said to be indicative of aneurism of the first part of the aortic arch, is obtained, according to Dr. Ewart, as follows: The patient being seated, the operator, standing behind him, places the tips of the index fingers under the lower edge of the cricoid cartilage, and gently raises it. The patient's head rests on the chest of the operator. The sign consists in a downward traction of the trachea, felt with each systole of the heart. It is not present in all cases of aneurism of the first part of the arch, nor is it pathognomonic of this condition.

C. OTHER ORGANS

A feeble pulsation may sometimes be felt in the thyroid gland with each systole of the heart.

Pulsation of the liver is best appreciated by means of palpation, and its expansile character should be carefully observed. The

possibility of transmitted pulsation from the right heart should be excluded before the existence of true pulsation is admitted.

The lower edge of the liver is frequently palpable in cases of hepatic enlargement.

Cardiac disease, as Dr. Head's brilliant investigations have shown, is attended by referred pain and tenderness over certain well defined areas of the chest and scalp. Thus affections of the aortic area and ventricle give rise to reflected pain and tenderness over the upper areas of the chest (2, 3, 4, 5 dorsal) and the mid-orbital region of the scalp. Lesions of the mitral area and auricles are attended by referred pain and tenderness, in the lower part of the chest and upper part of the abdomen (5, 6, 7, 8, 9, dorsal areas) and in the temporal and fronto-temporal areas of the scalp. The tenderness in these areas can be elicited by picking up the superficial structures between the thumb and finger, or by means of the blunt end of a pin.

SUB-SECTION III.

PERCUSSION

Two signs are elicited by means of percussion: the first and more important being the sound emitted by the part percussed, and the second, the degree of resistance, or the density of the spot that is struck.

Percussion is distinguished under the terms 'superficial' or 'deep,' according as the finger or pleximeter is struck gently or forcibly.

Percussion of the cardiac area will be considered under the following arrangement:—

A. HEART

(a) *Superficial cardiac dulness*

1. Increase of
2. Diminution of

(b) *Deep cardiac dulness*

1. Lateral increase of
2. Upward increase of
3. Downward increase of

B. PERICARDIUM

(a) *Dulness due to effusion*

(b) *Dulness due to adhesions*

C. GREAT VESSELS

D. OTHER ORGANS

A. HEART

(a) **Superficial Cardiac Dulness**

By superficial cardiac dulness is meant the extent of the heart's surface which is uncovered by the lung, and in contact with the chest wall. This area has already been marked out, under normal conditions, on the surface of the chest (see p. 6), and it is necessary only to add that light percussion should be used in defining its outline.

1. **Increase of:** The area of superficial cardiac dulness is increased by (a) deep expiration, (b) lying on the left side, (c) retraction of the lungs, (d) enlargement of the heart, and (e) pericardial effusion and pericardial adhesions.

2. **Diminution of:** The area of superficial cardiac dulness is diminished by (a) deep inspiration, (b) emphysema, (c) pneumo-pericardium, (d) displacement of the heart due to air in the pleural cavities, stomach, intestine, and abdominal cavity, or to pleuro-pericardial adhesions, which may drag the organ under the lungs, and possibly by (e) atrophy of the heart.

The information obtained by the superficial method of cardiac percussion is, on the whole, more useful in the diagnosis of diseases of the lungs than of the heart.

(b) Deep Cardiac Dulness

The area of deep cardiac dulness corresponds approximately with the size of the heart. The outline of the organ on the surface of the chest under normal conditions has already been mapped out (see p. 5) and need not be described again. In order to trace out the area in question deep percussion should be employed and a definite mode of procedure adopted.

Percussion, in all cases, whether superficial or deep, should be commenced well outside the normal limits of the dulness to be percussed, and the finger is then gradually advanced towards the line where resonance is impaired. In the case of deep cardiac dulness, for instance, the point where the lung resonance begins to be interfered with by the underlying heart should be marked on the chest wall, and the joining of the points, obtained by percussion from all sides, will represent the outline of the heart.

1. **Lateral increase of the area of deep cardiac dulness.**
Increase to the right: For clinical purposes the right limit of cardiac dulness, under normal conditions, is defined by the right edge of the sternum. Extension of dulness beyond this line means enlargement of the right auricle and *a priori* of the right ventricle. It will be noticed that the right limit of cardiac dulness as obtained by precussion does not exactly correspond with the outline of the right auricle as marked out on the chest wall (see diagram, p. 5). This is due to the fact that the outline of the heart is drawn from measurements in the cadaver, whereas in life it is probable that, owing to the pressure of the inflated right lung, and the tone of the auricular wall, the right auricle is situated rather more to the left than is represented.

Increase to the left: Extension of the cardiac dulness beyond the left vertical nipple line, which is the normal limit, indicates dilatation of the left ventricle.

Increase to the right and left: A bilateral increase of the cardiac dulness is indicative of enlargement of both sides of the heart, and is the condition that is commonly found.

2. Upward increase of the area of deep cardiac dulness. The upper limit of cardiac dulness, under normal conditions, is the level of the third rib. Extension of dulness above this line means, in the absence of pericardial effusion, enlargement of the infundibulum of the right ventricle, enlargement of the left auricle, aneurism of the aorta or mediastinal tumour.

3. Downward increase of the area of deep cardiac dulness. The downward extent of cardiac dulness is often difficult to define accurately, owing to the presence of the liver.

The lower limit of the left-sided portion of the organ can generally be made out, especially if the stomach is distended with gas, and the lower limit of the right-sided portion may often be approximately determined by a slight increase in the intensity of the dulness, and a feeling of increased resistance on passing from the heart to the liver.

When the lower limit of the heart cannot be determined by percussion, it may be nearly defined by drawing a line, slightly curved upwards, from the apex beat to the angle of junction of the upper limit of liver dulness with the right limit of cardiac dulness.

Extension downwards of the cardiac dulness means, on the left side of the heart, hypertrophy of the left ventricle, and on the right side of the organ, enlargement of the right ventricle.

An increase in the area of cardiac dulness downwards and to the left indicates hypertrophy and dilatation of the left ventricle.

B. PERICARDIUM

(a) Dulness due to Effusion

Effusion into the pericardial cavity causes an increase upwards of the præcordial dulness, which may reach as high as the cartilage of the first rib. It may extend downwards as low as the sixth rib or space, while laterally it may, with a copious effusion, stretch from the right vertical nipple line to the left axillary line.

The shape of the area of dulness is pyriform when the amount of effusion is slight, and pyramidal or triangular when it is extensive, with the base of the figure directed downwards in both cases. Owing to the pushing aside of the lungs by the enlarged pericardial sac, the line of dulness separating it from the pulmonary tissue is abrupt and well defined, and the finger experiences a sense of increased resistance over the effusion.

The relation of the apex beat to the area of dulness must be carefully ascertained.

With a copious pericardial effusion the apex beat lies well above and inside the area of dulness, that is to say the dulness due to the effusion extends below and outside the apex beat.

(b) **Dulness due to Adhesions**

Pericardial adhesions are often associated with a permanent increase in the area of cardiac dulness, which depends on enlargement of the heart.

C. GREAT VESSELS

Dulness due to dilatation of the ascending aorta may often be percussed out over the second right intercostal space and adjacent portion of the sternum, and appears as an extension upwards and to the right of the cardiac dulness. Aneurism of the vessel gives rise to a larger area of dulness in this situation.

Dulness over the upper sternal region and adjacent portion of the left side of the thorax is sometimes associated with aneurism of the transverse portion of the aortic arch.

Dilatation of the pulmonary artery gives rise to dulness in the second left interspace close to the edge of the sternum.

D. OTHER ORGANS

The size of the liver should always be determined by means of palpation and percussion in cases of morbus cordis. Normally the upward extent of the organ in the right vertical nipple line is the level of the sixth rib, though the upper surface of the liver, owing to its dome-shaped conformation, corresponds, in easy breathing, with a horizontal line drawn at the level of the base of the ensiform cartilage, or middle of the eighth dorsal vertebra. The downward extent of the liver in the nipple line is the costal margin, while in the median line it extends one and a half inches below the base of the xiphoid cartilage.

Enlargement of the liver is one of the earliest indications of interference with passage of blood through the right heart and lungs, hence the importance of a systematic examination of the organ in all cases of heart disease. Indeed, so sensitive is the liver to disturbance of the blood current through the right heart and lungs that it might almost be called "the barometer of the circulation."

SUB-SECTION IV

AUSCULTATION

Auscultation of the heart will be considered under the following scheme :—

A. HEART SOUNDS AND THEIR MODIFICATIONS

- (a) Position and direction of conduction*
- (b) Character*
- (c) Rhythm*
- (d) Reduplication*

B. ADVENTITIOUS SOUNDS*(a) Endocardial*

1. Organic murmurs
 - Physical causes of
 - Site of production and direction of transmission
 - Character
 - Rhythm
2. Hæmic murmurs
 - Causes of
 - Site of production and direction of transmission
 - Character
 - Rhythm

(b) Exocardial

1. Friction sounds
 - Pericardial
 - Pleural
 - Pleuro-pericardial
2. Murmurs
 - Cardio-pulmonary

C. VASCULAR SOUNDS

1. Normal sounds
2. Adventitious sounds
 - Arterial
 - Venous

A. THE HEART SOUNDS AND THEIR MODIFICATIONS

The normal sounds.—The method of production of the normal sounds of the heart has already been discussed (see p. 12), and does not require any further consideration.

Before describing the position in which the sounds are heard, a brief account will be given of the mode of procedure that should be adopted in auscultating the heart.

The chest piece of the stethoscope is placed firstly over the apex beat and the region of the chest wall immediately surrounding it, which may collectively be termed the "mitral area." The instrument is then gradually advanced, obliquely upwards and outwards, into the left axilla, and thence as far as the angle of the left scapula. It is then placed upon successive points on the chest wall, in a line connecting the apex beat with the second right costal cartilage, at its junction with the sternum. The second right costal cartilage is known as the "aortic cartilage," and this region of the chest wall, with the adjacent portion of the second interspace, is called the "aortic area." From here the stethoscope is carried upwards to the episternal notch and right side of the neck over the carotid artery,

and downwards along the right sternal edge. It is then placed close to the sternum over the second left costal cartilage, termed the "pulmonary cartilage," which with the adjacent portion of the second interspace is known as the "pulmonary area." The instrument is now carried along the left sternal edge to the base of the ensiform cartilage. The fourth and fifth left intercostal spaces, for about an inch to the left of the sternum, and the region of the chest wall immediately surrounding the base of the ensiform cartilage, are collectively termed the "tricuspid area."

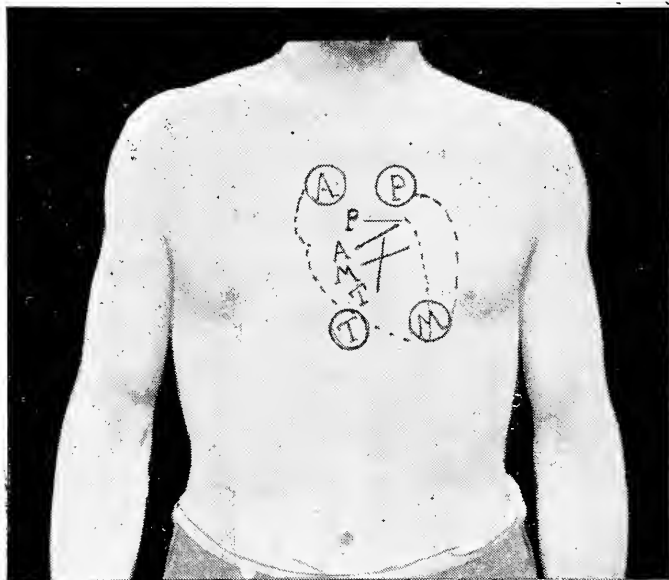


FIG. 6. THE AREAS IN WHICH THE SOUNDS PRODUCED AT THE VARIOUS CARDIAC ORIFICES ARE MOST DISTINCTLY HEARD

M (within circle)=mitral area ; A (within circle)=aortic area ; T (within circle)=tricuspid area
P (within circle)=pulmonary area

To distinguish between the two sounds of the heart, a finger should be put on the apex beat, or on the carotid artery in the neck, while the stethoscope is placed in the mitral area; the first sound is synchronous with the cardiac impulse or pulse wave, as the case may be.

(a) Position and Direction of Conduction of the Normal Heart Sounds

The left ventricle first sound.—The left ventricle first sound is most distinctly audible at and to the left of the apex beat. It is also heard with a variable degree of distinctness in the aortic area.

The aortic second sound.—The aortic second sound is heard best in the aortic area, but it is distinctly audible over the carotid arteries in the neck, especially on the right side, and in this situation is not liable to be confused with the pulmonic second sound. The aortic second sound can also be well heard at and to the left of the apex beat.

The pulmonic second sound.—The pulmonic second sound is heard most distinctly in the second left interspace close to the sternum. It is also plainly audible over the second left costal cartilage, and over the whole of the right ventricle. It is not normally audible at the apex of the heart.

The right ventricle first sound.—The first sound of the right ventricle is heard over the whole of the organ in relation with the chest wall, but it is most distinctly audible in the tricuspid area.

It will be noticed that the areas in which the various heart sounds are best heard do not necessarily correspond with the anatomical position of the structures producing them, and for the following reasons.

It has already been explained that the first sound of the heart is composed of a muscular and a valvular element. The muscular element of the first sound gives rise to no difficulty, as it is naturally heard most distinctly where the ventricles are in closest contact with the chest wall, and hence, in the case of the left ventricle, is most plainly audible at the apex of the heart.

The valvular element of the first sound of the left ventricle is not audible over the anatomical position of the mitral orifice, because here a considerable thickness of lung tissue, which is a bad conductor of sound, is interposed between the heart and chest wall. The valvular vibrations are, however, transmitted along the wall of the left ventricle, and are best heard at the apex of the heart, which, uncovered by lung, comes into close relation with the thoracic parietes.

For similar reasons the first sound of the right ventricle is most distinctly audible in the tricuspid area, and not directly over the site of the tricuspid valve. The left ventricle first sound is heard in the aortic area, and is conducted thither by the walls of the aorta.

With respect to the aortic second sound, it is heard most distinctly in the aortic area, where the vessel comes nearest to the surface. The vibrations due to the sudden tension of the semilunar valves are transmitted along the course of the aorta by means of the arterial wall and the contained column of blood. A similar mechanism explains the conduction of the aortic second sound into the neck, and it is transmitted to the apex of the heart by the wall of the left ventricle.

The reason that it is not heard over the site of the aortic orifice is that here the vessel is covered, not only by lung, but also by the infundibulum of the right ventricle, which interferes with the transmission of vibrations to the surface of the chest.

The pulmonary second sound is heard most distinctly over the exact anatomical position of the orifice, viz. at the upper border of the third left costal cartilage close to the sternum. The sound is transmitted upwards along the course of the vessel, as high as the second costal cartilage, and downwards by the wall of the right ventricle, to the base of the ensiform cartilage, and to within an inch of the apex beat.

(b) Character of the Normal Sounds of the Heart

The sounds of the heart are usually represented by the familiar syllables "lubb-dup," which correspond to the first and second sounds respectively. They convey the idea of sudden tension, and can be imitated by the more or less rapid stretching of longer and shorter pieces of string or membrane. The border of an ordinary pocket-handkerchief answers the purpose perfectly well.

The first sound is duller, longer, and louder than the second, which is short and sharp. Normally the left ventricle first sound is duller and longer than that of the right, which is relatively short and sharp. This difference is in all probability explained by the relative preponderance of the muscular element in the production of the left ventricle first sound.

The pulmonary second sound at the base of the heart is under normal conditions louder than the aortic, but, according to some observers, this statement is open to doubt. The relative intensity of the two sounds is apparently determined largely by the age of the individual (Cabot).

(c) Rhythm of the Normal Sounds of the Heart

The relative time duration of the events composing the cardiac cycle may be stated approximately as follows:—

The first sound occupies nearly three-tenths of a second. The interval between the first and second sounds one-twentieth of a second.

The second sound occupies one-tenth of a second. The long silence, or in other words the diastole of the ventricles, occupies five-tenths of a second.

Under normal conditions the relative lengths of the interval between the first and second sounds, and second and first, are preserved, though the rate of the heart beats per minute may vary within very wide limits.

Position and direction of conduction.—In order to avoid subsequent repetition, it may be stated generally that the degree of distinctness with which the sounds of the heart are heard depends not only on the character of the sound, but also on the thickness of the thoracic wall, and the extent to which the organ is overlapped

by lung tissue, or by other material, such as air, fluid, solid tumours, etc. For example, the first sound of the heart is more or less indistinct in cases of emphysema of the lungs, and of pericardial effusion or growths.

On the other hand, the sounds of the heart become more distinct in those conditions in which the organ comes nearer to the chest wall, as in retraction of the lungs, etc. Thus the pulmonary second sound may appear to be accentuated in retraction of the left lung, and similar effects are observed as regards the other sounds of the heart, under like circumstances.

These conditions are mentioned in order that due allowance may be made for them in estimating the character of the various cardiac sounds.

The left ventricle first sound.—In hypertrophy of the left ventricle, and in cases of high systemic tension, the first sound of the heart becomes less distinct, and may be quite inaudible in the aortic area.

On the other hand, it becomes more distinct in this situation, in conditions of low arterial tension and in dilatation of the left ventricle. Apparently the character of the left ventricle first sound largely influences the degree of its conduction along the aorta.

The aortic second sound.—The aortic second sound becomes inaudible at and to the left of the apex beat, when, owing to enlargement of the right heart, the left ventricle, and with it the apex, is displaced from its normal position. This occurs in cases of mitral stenosis, though probably in this disease the weakness of the second sound also affects its conduction.

The absence of the aortic second sound over the carotid arteries in the neck depends on damage to the semilunar valves, the result of injury or disease, whereby these structures are unable to offer sufficient check to the backflow of blood towards the ventricle to produce vibrations of the aortic walls.

It will be seen, therefore, that the absence of the second sound in the neck is indicative of a considerable amount of regurgitation into the left ventricle.

The pulmonic second sound.—This sound, in cases of enlargement of the right heart, may be heard as far to the left as the normal position of the apex beat, and is transmitted there by the walls of the right ventricle, which has usurped the place of the left.

In some instances of consolidation of the left upper lobe of the lung, the pulmonary second sound may be heard over a large area of the left chest.

The right ventricle first sound.—In cases of enlargement of the right ventricle, the first sound may be heard further to the left than usual.

Modifications in the character of the sounds of the heart.—Three distinct elements are to be distinguished in every tone, viz. :—

1. Intensity (loudness)
2. Pitch
3. Quality (timbre)

The three elements taken together determine the character of a tone. With regard to the sounds of the heart, which are not true musical tones, it is often difficult to state with precision the exact modifications that they may undergo. This statement applies more particularly to changes in quality, but the attempt will be made to state, so far as possible, in technical language the variations to which the different sounds of the heart are liable.

Left ventricle first sound.—Under normal conditions the variations in character of the left ventricle first sound are comparatively unimportant, and consist mainly in alterations in duration and intensity.

The first sound is short and loud when the ventricle is acting rapidly and the arterial tension is low, as may occur in nervous conditions, for instance in Graves' disease, or in the early stages of fever.

Conversely, it is long and diminished in intensity when the ventricle is contracting slowly against increased systemic tension.

In hypertrophy of the ventricle the muscular element becomes obtrusive, and the first sound is therefore prolonged and dull. Stated in technical language, the sound is characterised by increased duration, diminished intensity, and slightly altered quality.

In dilatation of the ventricle the condition of affairs is reversed. Here the valvular element becomes prominent, and the sound is short, sharp, and loud. In other words, the sound is characterised by diminished duration, by heightened pitch, and by increased intensity. The quality of the sound also undergoes slight modification by reason of the change in the mechanism of its production.

Shortness of the left ventricle first sound, with diminution of intensity, which may go on to complete extinction, is indicative of extreme weakness of the ventricular walls, and occurs in prolonged febrile conditions, such as enteric fever and in other diseases productive of protracted malnutrition of the myocardium.

In mitral stenosis the first sound of the left ventricle is remarkably and characteristically short, sharp, and loud, and at the same time altered in quality. In cases of mitral regurgitation, the first sound may be partially or wholly replaced by the systolic murmur.

The aortic second sound.—The most common variation affecting this sound is a change in intensity, which becomes either relatively or absolutely increased or diminished.

An increase in the intensity of the sound produced at the aortic or pulmonic orifices is known under the name of accentuation.

Accentuation of the aortic second sound occurs in high arterial tension with a forcibly acting left ventricle. It may also occur in

association with low arterial tension, if the ventricular systole is vehement; but in such cases the accentuation is not relatively apparent, owing to the concurrent accentuation of the pulmonic second sound.

Accentuation of the aortic second sound is almost invariably produced by dilatation or aneurism of the ascending aorta, and in such conditions the sound is not only accentuated but is also of markedly lower pitch.

A further modification of the second sound is observed when, in association with dilatation of the vessel, the walls of the aorta have undergone thinning, and more or less fusion of the three coats of the artery has taken place. In addition to an alteration in intensity and pitch, the sound now acquires a change of quality to a more musical tone, which is highly characteristic of the affection in question, though a similar modification may be produced by aneurism of one of the sinuses of Valsalva.

Diminution in intensity, or feebleness of the aortic second sound, is associated with weakness of the left ventricle due to myocardial inflammation or degeneration. The second sound is enfeebled also in mitral stenosis and in nearly all forms of disease affecting the aortic valve.

Weakness or disappearance of the aortic second sound in the neck in cases of aortic incompetence is significant of a considerable amount of valvular insufficiency.

The pulmonic second sound.—The pulmonic second sound is accentuated in all conditions which give rise to obstruction to the passage of blood through the lungs. It occurs therefore in affections of the lungs such as pneumonia, bronchitis, emphysema, pleural effusion, etc., and sooner or later in all diseases of the left side of the heart.

Weakness of the pulmonic second sound is of importance only when observed in conjunction with previous accentuation. With this sequence of events, enfeeblement of the sound is indicative of dilatation of the right ventricle with leakage through the tricuspid valve.

The right ventricle first sound.—So long as the myocardium remains healthy, the right ventricle first sound is intensified in all conditions which interfere with the normal circulation of blood through the lungs. The circumstances under which pulmonary obstruction occurs have been referred to under the causes of accentuation of the pulmonic second sound.

Enfeeblement of the right heart first sound occurs as the result of myocardial inflammation or degeneration, and in the later stages of dilatation of the ventricle from any cause.

Modifications in the rhythm of the sounds of the heart.—For clinical purposes alterations in the relative time duration of the various events composing the cardiac cycle may be considered to take place in two directions, with the result that the sounds of the heart are spaced (that is, separated by a longer interval than normal) or approximated.

The first effect may be produced either by prolongation of the interval between the first and second sounds, or by the shortening of the diastole; the second, by the shortening of the interval between the first and second sounds, or by prolongation of the diastole.

Spacing of the sounds of the heart may be carried to the point at which they become equidistant, and this condition has been termed *embryocardia*, or the tick-tack action of the heart, from its resemblance to the rhythm of the foetal heart sounds, or to the ticking of the pendulum of a clock, or of a watch.

Equidistance of the two sounds of the heart, from prolongation of the interval between them, is found most commonly in association with high arterial tension, and is then indicative of impending failure of the left ventricle.

Thus, in these cases, the ventricle, in spite of the increased resistance to the discharge of its contents, is still just able to empty itself, but it does so with much greater difficulty, and therefore more slowly than usual; hence the spacing of the sounds.

A similar phenomenon may be observed in enteric fever as the result of myocardial degeneration, and is then usually of fatal import.

Equidistance of the sounds of the heart is sometimes associated with tachycardia, and in such instances is produced by the shortening of the diastole.

Approximation of the first and second sounds of the heart usually indicates weakness of the left ventricle, consequent on myocardial disease, or high arterial tension. Here the ventricle does not complete its contraction by reason of the absolute or relative inability of its muscular walls to maintain the intra-ventricular pressure above that in the aorta throughout the systole. So soon, therefore, as the pressure in the aorta exceeds that in the ventricle the semilunar valves close, and the second sound occurs. This event may take place at any stage of the ventricular contraction, with a corresponding variation in the degree of approximation of the sounds. In some instances the second sound follows the first almost immediately. The phenomenon is observed with failure of the heart in diphtheria and other acute febrile conditions, and in the early and late stages of kidney disease. The approximation of the sounds, under these circumstances, is of very grave significance.

Approximation of the sounds of the heart may also be heard in some cases of low arterial tension with a rapid action of the organ,

such as occurs in febrile and emotional states, and is then of no prognostic value.

(d) Reduplication of the Sounds of the Heart

Either the first or second sound of the heart may be reduplicated. The reduplication usually implicates one sound at a time, but occasionally both are affected together. The two elements composing the double sound may be closely approximated, or they may be separated by an appreciable interval, producing, in the first event, an effect resembling the double beat upon a drum, and hence called the "*Bruit de rappel*," and in the second, a likeness to the footfall of a horse at a canter, the "*Bruit de galop*."

Thus in the case of the doubling of the first sound of the heart the result is heard as "*lurrub-dup*" or "*lublub-dup*," and of the second, as "*lub-durru*" or "*lub-dupdup*," according to the degree of separation of the two elements of the double sound.

There is no kind of agreement among writers on the subject, either as to the cause, significance, or even area of greatest audibility of the phenomena in question, and it will be possible to give but a brief outline of the views which have been advanced to explain them.

Reduplication of the first sound of the heart.—Reduplication of the first sound is usually most distinctly audible just internal to the apex beat, and along the line of the interventricular septum, but it may also be heard in the tricuspid area, and along the left edge of the sternum as high as the third rib. It is rarely, if ever, well heard over the base of the heart.

Reduplication of the first sound occurs most commonly in association with hypertrophy of the heart, the result of high arterial tension, such as obtains in some forms of kidney disease and in other disorders.

It is also observed in cases of dilatation of the heart following myocardial disease, or pericardial adhesion, and it may occur in the absence of any appreciable cardiac lesion (Potain).

Reduplication of the first sound is probably of little or no clinical significance, except when it is found in association with high arterial tension, particularly of nephritic origin, and it is then a sign of commencing dilatation of the left ventricle from failure.

The doubling of the first sound of the heart has been ascribed to a want of synchronism in the contraction of the two ventricles. Under normal conditions the relation between the intra-ventricular pressure and the muscular power of the ventricles is so adjusted that the two sides of the heart contract simultaneously. It is supposed that an increase of intra-ventricular pressure occurring on either side of the heart might, if sufficiently great, upset the normal balance and cause asynchronism. Thus the ventricle work-

ing against the increased pressure might contract later (according to Dr. Barr earlier) than usual, and especially may this be the case if, as Sir William Broadbent supposes, the ventricle affected is beginning to dilate before the resistance that has to be overcome.

Against this view has been advanced the fact that physiologists have never observed asynchronism in the contraction of the right and left sides of the heart. It is, however, extremely likely that under physiological conditions of the circulation this observation may hold good, but it is no argument against the view that asynchronism might occur under pathological influences.

The reduplication of the sound is most distinctly audible along the course of the interventricular septum, and in some instances is palpable in this situation. Moreover, in cases where doubling of the first sound is observed, the two elements composing it can be heard when the cusps of a differential stethoscope are placed over the right and left ventricles, where on separate examination, but one sound is audible (Broadbent). It would therefore appear highly probable that a want of synchronism in the contraction of the two ventricles is a cause of reduplication of the first sound of the heart.

The explanation that has just been given may not, however, cover all the cases in which reduplication of the first sound of the heart is heard. The phenomenon was ascribed by Dr. G. Johnson to an audible contraction of the auricle immediately preceding the ventricular systole. It is *a priori* improbable that the auricular contraction could give rise to a sound, and further, the area of audibility of the double sound as described by Dr. Johnson, and on which his view was largely based, is contrary to the experience of most observers.

Dr. Sansom attributes all forms of reduplication of the sounds of the heart to tension of the curtains of left auriculo-ventricular valve during some part of the diastole of the ventricle. Thus, in the present instance, he supposes that a forcible auricular contraction causes an impulse to the apex of the left ventricle, which by *contre-coup* gives rise to vibrations of the mitral curtains, and the sound thereby produced is heard just before the ventricular first sound.

There is, however, no cause in most of the cases in which reduplication of the first sound occurs for a forcible contraction of the auricle, since the stress of the circulation falls on the ventricle; hence the explanation given by Dr. Sansom cannot be regarded as of general application.

Reduplication of the second sound of the heart.—Reduplication of the second sound is usually most distinctly heard over the base of the heart, and at the left edge of the sternum about the level of the third intercostal space. According to Dr. Sansom it is most plainly audible just inside the apex beat, and along the left edge

of the sternum, at the level of the junction of the fourth and fifth costal cartilages. There is no doubt that an apparent doubling of the second sound of the heart is sometimes audible immediately to the inner side of the apex beat and not at the base, in cases in which a presystolic murmur subsequently appears, but the mechanism of its production differs from that of the reduplication previously mentioned.

Reduplication of the second sound of the heart occurs most commonly in mitral stenosis, but it may be observed with pulmonary obstruction from any cause. It is also heard in pericarditis and myocarditis, and in high arterial tension with hypertrophy of the left ventricle.

Except when it is heard at the apex of the heart and not at the base, reduplication of the second sound is of no clinical significance. It has been ascribed to a want of synchronism in the closure of the semilunar valves on the two sides of the heart. The asynchronism may be caused by increased systemic or pulmonic tension, which delays the completion of the systole of the corresponding ventricle, and consequently the closure of the semilunar valves.

This explanation, while accounting for the reduplication of the second sound as heard at the base of the heart, does not elucidate the apparent doubling of the second sound audible at the apex only.

This is probably due, as Dr. Sansom suggests, to tension of the segments of the left auriculo-ventricular valve, which is caused by a more rapid flow of blood than usual into the ventricle during the early part of the diastole consequent on increased pressure in the auricle.

Apparent doubling of the second sound which is heard in the neighbourhood of the apex beat only is, therefore, a sign of considerable diagnostic importance, since it is almost invariably the precursor of a presystolic murmur, indicative of stenosis at the mitral orifice.

B. ADVENTITIOUS SOUNDS

(a) Endocardial

• Audible vibrations, exclusive of the normal heart sounds and their modifications, accompanying the cardiac movements, and generated as the result of morbid conditions of the heart and pericardium, are known as murmurs.

Such superadded or adventitious sounds may partially or wholly replace the normal sounds of the heart.

For clinical purposes murmurs may be divided into—

- (a) Endocardial murmurs
- (b) Exocardial murmurs
- (c) Vascular murmurs

Endocardial murmurs may be further subdivided into—

- (a) Organic murmurs
- (b) Functional or Hæmic murmurs

1. **Organic murmurs.**—A murmur which is due to the presence of an appreciable morbid lesion is known as an organic murmur.

Physical Causes of Organic Murmurs

A murmur is the sound produced, under certain conditions, by the passage of gas or fluid along a tube. So long as the tube has a smooth internal surface, and does not alter in calibre, the circulation of fluid through it does not give rise to any audible vibrations. If, however, a constriction be introduced at any point along the course of the tube a “fluid vein” is formed immediately beyond the narrowed portion, which gives rise to sonorous vibrations that may be heard as a murmur. In the case of the heart the size of the various cavities and orifices is so adjusted that, under normal conditions, no sound is produced by the circulation of blood through them. Whenever a cavity or orifice is increased or diminished in size, either relatively or absolutely, the conditions for the production of a fluid vein are present, and consequently a murmur may be heard.

So far as the clinical expression of a murmur is concerned, it must be borne in mind that the vibrations in the blood stream caused by the formation of a fluid vein, though sonorous, are in all probability largely modified by the conducting properties of the surrounding solid structures, by means of which they are transmitted to the surface of the chest, and hence to the ear.

The presence of a fibrous cord or shred of membrane capable of free vibrations in the course of the blood stream may give rise to a musical murmur. Such conditions may exist in the heart when, in consequence of the improper adaptation of the margins of the valves guarding an orifice, the free edges of the valvular curtains can vibrate, or when a shred of fibrin, formed as the result of some morbid process, hangs loose in the blood stream.

A musical murmur can also be produced, under certain conditions, by the vibrations of a fluid vein alone. The loudness of a murmur depends for the most part on the swiftness of the flow producing it, and this is determined by (a) the *vis a tergo*, (b) the quality of the circulating fluid, and (c) the degree of constriction of the tube. If the velocity of the flow falls below a certain rate no murmur is produced.

A murmur is propagated in the direction of the flow, and is most distinctly heard immediately to the distal side of the site of its production.

This statement holds good with respect to the heart, but the area of audibility of a cardiac murmur is greatly modified by the various

solid structures along which the sonorous vibrations are conducted to the surface of the chest wall.

Cardiac murmurs are distinguished as (*a*) direct or obstructive, or (*b*) indirect or regurgitant, according as they are produced in the course of or against the natural direction of the blood stream.

Obstructive murmurs are due to—

1. The narrowing of an orifice, the result of a local constrictive process; or an impediment to the onward flow of blood through an opening by reason of an outgrowth or projection from its valve, or from adjacent structures.
2. The narrowing of an orifice relatively to the size of the channel situated immediately in front of or behind it. In such cases the dimensions of the orifice remain unchanged, while the cavity or lumen of the structure in immediate relation with it, either anteriorly or posteriorly, becomes dilated.

Regurgitant murmurs are due to—

1. The defective closure of a normally closed orifice, the result of disease affecting the valve that guards it.
2. The dilatation of an orifice, such as the auriculo-ventricular, in consequence of the relaxation and stretching of its muscular girdle, so that the curtains of its valve are unable to come into complete apposition.
3. The dilatation of an orifice owing to its involvement in a like process affecting primarily the channel beyond it, with the result that the proper adaptation of the valvular segments becomes impossible.

Murmurs are also distinguished, according to the period of their occurrence in the cardiac cycle, as (*a*) systolic or (*b*) diastolic.

A murmur that is heard between the beginning of the first sound and the occurrence of the second is called "systolic," while one which appears between the beginning of the second sound and commencement of the first is called "diastolic."

Diastolic murmurs are subdivided into—

1. Murmurs occupying the whole diastole.
2. Murmurs occupying a portion only of the diastole, to wit—
 - (*a*) Early diastolic or post systolic
 - (*b*) Mid-diastolic
 - (*c*) Late diastolic or presystolic

The relation of murmurs to the different periods of the cardiac cycle may be diagrammatically represented as follows :—

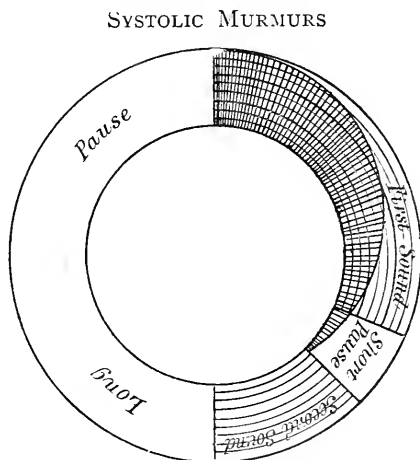


FIG. 9. DIAGRAMMATIC REPRESENTATION OF A SYSTOLIC MURMUR

It will appear, therefore, that systolic murmurs, due to organic disease, and produced at the arterial orifices of the heart, are indicative of obstruction to the blood stream, whereas at the auriculo-ventricular openings they are significant of regurgitation.

Conversely, diastolic murmurs originating at the arterial orifices indicate regurgitation, and at the auriculo-ventricular openings, obstruction.

Site of Production and Direction of Transmission of Organic Cardiac Murmurs

As a general rule a murmur is most distinctly heard over that portion of the chest wall which is nearest to the site of its production. The relation of the cardiac orifices to the thoracic parietes has already been defined under the anatomical description of the heart.

The deviations from the general rule just mentioned are numerous, and depend, for the most part, on the influence exerted by the conducting properties of (*a*) the solid structures interposed between the site of production of the murmur and the surface of the chest, and of (*b*) the blood current, or, as it usually termed, convection.

The modifications in the area of audibility of a murmur, thus produced, will be illustrated in the following account of the subject.

MITRAL ORIFICE.—Murmurs which are due to lesions at the left auriculo-ventricular opening may be either systolic or diastolic,

DIASTOLIC MURMURS

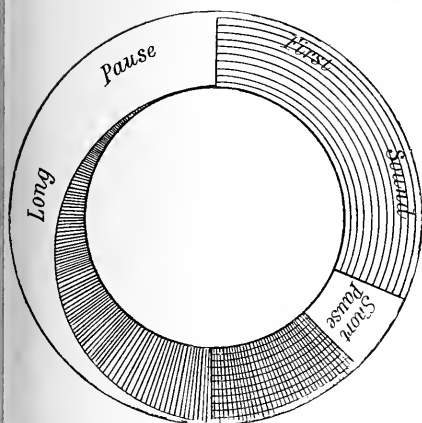


FIG. 10. DIAGRAMMATIC REPRESENTATION
OF AN ENTIRE DIASTOLIC MURMUR

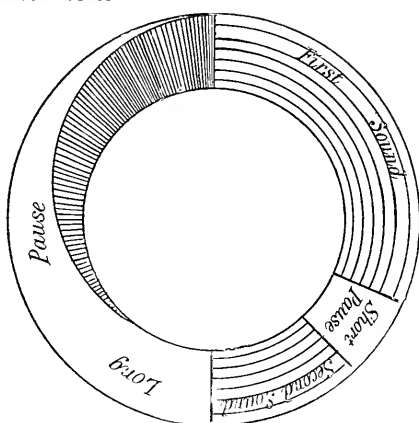


FIG. 11. DIAGRAMMATIC REPRESENTATION
OF A PRESYSTOLIC MURMUR

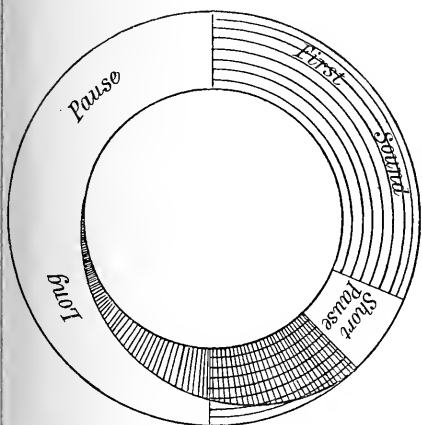


FIG. 12. DIAGRAMMATIC REPRESENTATION
OF AN EARLY DIASTOLIC MURMUR

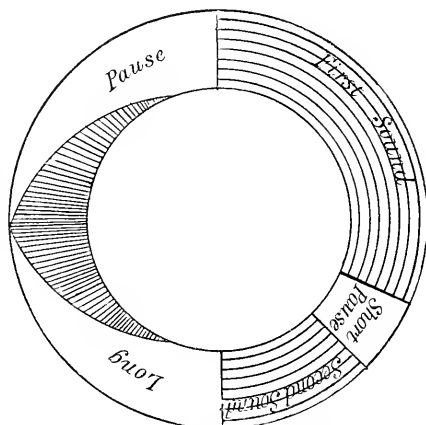


FIG. 13. DIAGRAMMATIC REPRESENTATION
OF A MID-DIASTOLIC MURMUR

the former indicating regurgitation through, the latter obstruction at the orifice.

Systolic mitral murmurs.—These are most distinctly heard in the mitral area, inasmuch as they are conducted thither by the wall of the left ventricle. They are also transmitted (under the influence of convection) for a variable distance into the axilla, and may be audible as far outwards as the angle of the left scapula. Systolic mitral murmurs are occasionally heard as far inwards as the left sternal edge, in the tricuspid area, and more rarely they are conducted upwards as high as the third or second rib. In exceptional instances systolic mitral murmurs are audible in the second and third left intercostal spaces, about two inches from the sternal border, and in this situation only.

Diastolic mitral murmurs.—All the varieties of diastolic murmur that were previously enumerated may originate at the mitral orifice. Mitral murmurs which occupy the whole diastole (or, for that matter, any part of it), entire diastolic murmurs as they have been called by Dr. Bristowe, have this peculiarity that they invariably *follow* the second sound. It is in this particular that they differ from aortic diastolic murmurs, which, whether audible in the mitral area or elsewhere, *accompany* and partially or wholly replace the second sound.

Mitral diastolic murmurs are usually most distinctly audible in the mitral area, and for the reason that they are conducted thither by the wall of the left ventricle.

The presystolic murmur, which, for the sake of convenience and on account of its importance, will be considered first, is best heard immediately above and to the inner side of the apex beat, and its area of audibility is limited to this situation with remarkable constancy. It is occasionally audible as far to the right as the left sternal edge and upwards as high as the third rib. Rarely, too, it is conducted into the axilla, and outwards as far as the angle of the left scapula.

The area of audibility of entire, early, and mid-diastolic murmurs corresponds, in the main, with that just described, though it is not usually so localized.

Combined systolic and diastolic mitral murmurs.—Cases of pure mitral obstruction are comparatively rare, since, by reason of the altered condition of the orifice and its valve, the segments of the latter are seldom able to come into effective apposition, and consequently a variable, though small, amount of leakage takes place.

The systolic murmur, produced by this means, is heard in the mitral area, but it is seldom well conducted into the axilla, and is rarely if ever audible at the angle of the left scapula.

The area of audibility of the accompanying presystolic bruit does not differ from that previously described for such murmurs.

AORTIC ORIFICE.—Murmurs originating here may be either systolic or diastolic, the former indicating obstruction at the orifice, dilatation of the aorta, or roughening of its lining membrane or of the cusps of the valve, the latter, regurgitation through the opening.

Systolic aortic murmurs.—Systolic murmurs due to obstruction at the orifice are heard best in the aortic area, and are well conducted upwards (under the influence of convection) into the neck, especially on the right side. They are also audible over the upper third of the sternum, since bone is a good conductor of sound.

Murmurs occasioned by atheromatous disease of the valve or aorta are heard in the same situations, but they tend to spread laterally rather than upwards.

Diastolic aortic murmurs.—The area over which these murmurs may be heard is a very large one. A diastolic aortic murmur is most distinctly audible at one time in the aortic area, at another in the mid-sternal region about the level of the fourth rib, in the mitral area, or over the base of the ensiform cartilage. It is usually well conducted downwards along the right and left edges of the sternum, and along an oblique line leading from the second right costal cartilage, at its junction with the sternum, to the apex beat.

A diastolic aortic murmur is sometimes audible at the apex only, and in such cases may closely resemble the presystolic murmur that is associated with mitral stenosis. The reason for this will be considered under the heading of the valvular lesion in question.

An aortic diastolic murmur may also be heard in the neck over the carotid arteries, and most distinctly on the right side.

Combined systolic and diastolic aortic murmurs.—The two murmurs are observed over their respective areas of audibility, as above described. In those situations where the areas of audibility overlap a "to and fro" murmur is heard.

PULMONARY ORIFICE.—Murmurs due to structural disease at this orifice, apart from congenital conditions, are rare. Nevertheless systolic and diastolic murmurs, indicating stenosis of the opening and incompetence of the valve respectively, are occasionally observed.

Systolic pulmonary murmurs.—These are heard most distinctly in the second left interspace close to the sternum, and are transmitted upwards and to the left for a short distance. They may also extend to the right as far as the aortic area on a level with the second interspace. They are inaudible in the neck.

Diastolic pulmonary murmurs.—These are, as a rule, most plainly audible in the pulmonic area, and are conducted downwards along the left sternal edge as far as the base of the ensiform cartilage. They may be heard over any portion of the right ventricle.

TRICUSPID ORIFICE.—Murmurs produced at this orifice may be either systolic or diastolic, and have, *mutatis mutandis*, the same significance as those heard at the mitral opening.

Systolic tricuspid murmurs.—These are heard most distinctly in the tricuspid area, and are conducted by the right ventricle to the right, and upwards along the sternal edge as high as the fourth interspace.

Diastolic tricuspid murmurs.—A presystolic murmur, indicating tricuspid stenosis, is sometimes heard at the left sternal edge, about the level of the fourth and fifth costal cartilages, and over the base of the ensiform cartilage.

Combined systolic and diastolic tricuspid murmurs.—A systolic and presystolic murmur are occasionally heard together in the tricuspid area.

Associated murmurs.—It not infrequently happens that more than one orifice of the heart is the seat of disease at the same time, and in such an event murmurs with different sites of production may coexist.

The most common association is that of systolic and diastolic aortic murmurs, with a systolic mitral bruit, and then either of the former with the latter. Combined systolic and diastolic murmurs produced at the aortic and mitral openings may also coexist, and more rarely still combined or single aortic murmurs may be associated with a presystolic mitral bruit. A systolic or presystolic mitral murmur, or a combination of the two, is not uncommonly associated with a systolic murmur of tricuspid origin.

Apart from congenital conditions, a presystolic tricuspid murmur is almost invariably associated with a mitral presystolic bruit.

With respect to the association of murmurs which are produced on the left and right sides of the heart, it should be noted that the relation of the lesions giving rise to them is nearly always one of cause and effect.

The Character of Organic Murmurs

Cardiac murmurs are described as blowing, rasping, purring, rumbling, etc., according to the fancy of the observer.

These terms apply, of course, to the quality of the sound, which is of little clinical importance.

Systolic mitral murmurs are usually soft and blowing, and sometimes possess a musical character, whereas systolic aortic murmurs are commonly rough and rasping.

The presystolic bruit due to mitral obstruction is peculiarly and characteristically rough and rumbling, and increases in intensity up to the first sound, at which it abruptly terminates. Presystolic tricuspid murmurs exhibit similar qualities.

Diastolic murmurs produced at the aortic orifice are usually soft and blowing, and they gradually diminish in intensity from their commencement with the second sound to their termination. They do

not, however, always possess these characters, for, as has already been pointed out, a murmur due to aortic regurgitation is sometimes audible at the apex only, and may then be indistinguishable, so far as its character is concerned, from that associated with mitral obstruction.

The distinctness with which a cardiac murmur is heard depends for the most part on posture, on the quantity and conducting quality of the material interposed between the site of production of the murmur and the ear, and on the loudness of the murmur.

Unless considered in the light of previous observations, the loudness of a murmur is of little or no diagnostic or prognostic value. It depends mainly on the swiftness of the blood current, and in the case of the heart this will be determined by the force of the auricular or ventricular contraction, as the case may be, and by the condition of arterial tension.

Speaking generally, a loud murmur is of less import than a soft one, as it is an indication that the heart is acting forcibly.

Gradual increase in the intensity of a murmur is a good prognostic sign, in so far as it is indicative of improving cardiac vigour. On the other hand, sudden or gradual decrease in the intensity of a murmur is often significant of serious failure of the power of the heart.

A soft murmur is, however, compatible with very slight damage either to the myocardium or endocardium.

The subject will be again referred to under the general account of the prognosis in valvular affections of the heart.

The Rhythm of Organic Cardiac Murmurs

It has already been pointed out that, with regard to their position in the cardiac cycle, murmurs may be either systolic or diastolic. Their time relations should be gauged clinically by reference to the sounds of the heart, to the impulse of the organ, and to the carotid pulse, which for all practical purposes is synchronous with the ventricular systole.

It must be borne in mind that palpation of the apex beat does not always afford reliable information as to the time of the contraction of the ventricles, as may be demonstrated in some cases of aortic regurgitation.

With reference to the position of murmurs in the cardiac cycle, two additional points remain to be mentioned, viz. (1) the relation of the murmur to the sound of the heart with which it is associated, and (2) the duration of the murmur.

Thus, by way of illustrating the first point, a systolic murmur produced at the mitral orifice may wholly or partially replace the first sound of the heart.

If the murmur wholly obscures the first sound, the indication is that there is an excessive production of sonorous vibrations at the orifice, or that the valvular segments are unable to undergo sufficient tension to produce a sound. Under either supposition the inference

is that the amount of regurgitation must be considerable. If, on the other hand, the murmur follows and only partially obscures the sound, it is obvious that the segments of the auriculo-ventricular valve are able to come together, but are unable to remain in apposition, which argues but a slight amount of leakage through the opening.

Again, a diastolic murmur produced at the aortic orifice may wholly or partially replace the second sound. If the second sound is inaudible it means that the valvular cusps fail to offer any effective check to the backflow of blood, and therefore that there must be a large amount of regurgitation. If, on the other hand, the murmur follows the second sound, or only slightly obscures it, the valve must still offer a hindrance to the regurgitant current, and consequently the leakage through the opening can be but slight.

These considerations, while affording a valuable means of estimating the degree of incompetence of a valve, are not absolute, and they must be weighed in conjunction with the other indications of the extent of the valvular insufficiency.

The duration of a murmur is of little clinical significance.

A long murmur, which depends on valvular incompetence, frequently indicates slight disease and a forcible action of the heart. On the other hand, a short murmur is sometimes a sign of serious failure of the cardiac power.

2. Hæmic murmurs.—The method of production of hæmic or functional murmurs (as they are sometimes termed) has long been a subject of controversy, and still remains undetermined. There can be little doubt, however, that the production of a hæmic murmur depends, as in the case of an organic murmur, on the formation of a fluid vein, which is sonorous. If the conditions under which a fluid vein may be formed can be shown to be present in anæmia, there is no necessity to endeavour to explain the occurrence of hæmic murmurs on other grounds. An attempt will be made to show that such conditions may exist, but it will also be necessary to briefly review the other chief theories which have been advanced to explain the formation of these murmurs.

The order in which hæmic murmurs appear in anæmia is usually as follows :—

1. The venous hum or "bruit de diable" in the neck.
2. A systolic murmur in the pulmonic area.
3. A systolic murmur in the mitral area.
4. A systolic murmur in the tricuspid area.
5. A systolic murmur in the aortic area.

Site of Production and Direction of Transmission of Cardiac Murmurs
due to Anæmia

The venous hum which may be heard in the neck in cases of anæmia, will be considered under its appropriate heading.

Cardiac murmurs of hæmic origin will be taken in the order of their usual appearance.

Pulmonic orifice.—A systolic bruit, which is most distinctly audible in the second left intercostal space close to the sternum, or, in other words, in the pulmonic area, is the earliest and most constant of the cardiac murmurs that are found in association with anæmia.

The area of audibility of the murmur as given here is not admitted by all writers on the subject, nevertheless the majority are agreed that the bruit appears soonest, and is heard best, in this situation. It is in all probability produced at the pulmonic orifice, and for the following reasons.

In anæmia, from whatever cause, there is present a general muscular malnutrition, and the heart suffers in common with the rest of the contractile tissues. Owing to the comparative thinness of its walls, the right ventricle feels the effect of this nutritional impairment at an early period of the disease, and a variable degree of dilatation of the ventricular cavity ensues. Consequently the orifice of the pulmonic artery, which by reason of its dense fibrous girdle remains unaltered in size, becomes relatively constricted as regards the cavity of the ventricle, and hence the conditions for the production of a fluid vein are present. This view, too, would explain the presence of a murmur, even if, as Duroziez asserts, the heart and its cavities are diminished in size in anæmia, for then the pulmonic orifice would be relatively dilated. It is possible, indeed probable, that the alteration in the quality of the circulating fluid may also be a factor in the production of the fluid vein. A fall of blood pressure in the aorta or pulmonary artery as the case may be is also a contributory factor in the production of a functional systolic murmur at the orifices of these vessels.

Balfour considers that the murmur under consideration is due to mitral regurgitation, which causes a vibration of the walls of the left auricular appendix, and therefore that the murmur is heard most distinctly over the position of the appendix, *i.e.* in the second left intercostal space one and a half to two inches outside the left sternal edge. Apart from the fact that the area in which the murmur is most plainly audible is not located by the majority of observers in the situation described by Balfour, it has been shown that in a large number of cases of anæmia the left auricular appendix does not come into contact with the chest wall.

Moreover, it is difficult to suppose that the small amount of regurgitation which takes place could cause any audible vibration of the auricular walls, seeing that in structural disease of the mitral valve, with considerable leakage, systolic murmurs are rarely heard in the situation described by Balfour.

Russell's theory, which accounts for the murmur by the pressure of a dilated auricle on the pulmonary artery causing a local narrowing of the vessel and hence a fluid vein, is equally improbable. There is no proof that the auricle is distended in anæmia, and furthermore, there is direct evidence that when the auricle is dilated, as in mitral stenosis, no pressure is exerted on the pulmonary artery, or at all events not sufficient to produce a murmur.

Chauveau supposes that in anæmia the total quantity of blood in the body is diminished, and this results in a partial collapse of the capillaries, small and medium-sized arteries and veins, and of the heart and its orifices. The aorta and pulmonic artery, owing to the anatomical structure of their walls, are unable to accommodate themselves to the altered circulatory conditions, and consequently they become relatively dilated as regards their respective orifices, and thus the conditions for the production of a fluid vein are produced.

It has not been proved that the total quantity of blood is diminished in anæmia; moreover, partial collapse of the orifices of the aorta and pulmonic artery would seem as difficult of accomplishment as of the vessels themselves.

According to Hayden, two factors enter into the causation of hæmic murmurs, viz. (1) friction of the blood corpuscles against one another and against the edges of the cardiac orifices and walls of the vessels, and (2) vibrations of the heart and walls of the vessels.

It is by no means certain that increased friction between the blood corpuscles, etc., could give rise to sonorous vibrations. Moreover, if this were possible, the occurrence and intensity of the bruit thereby produced should be regulated by the degree of anæmia, which is not the case with respect to hæmic murmurs.

Explanations of the mode of formation of hæmic murmurs based on neuro-muscular causes, such as those advanced by Dr. Sansom, can hardly be considered satisfactory in the present state of our knowledge of these conditions.

Foxwell (Bradshaw Lecture, 1899) contends that the systolic murmur heard in the second and third intercostal spaces in cases of cardiac debility (due to anæmia and other causes) depends on a localized dilatation of the conus arteriosus and pulmonary artery. He states that dilatation of the conus arteriosus and pulmonary artery is constantly found post mortem in patients who during life exhibited this functional pulmonary murmur, and he advances the following arguments with the object of showing that this association is one of cause and effect:—

1. The pulmonary orifice is not increased in size, and therefore becomes relatively constricted as regards the enlarged conus,

with the consequent production of the necessary conditions for the formation of a fluid vein and hence of a murmur.

2. The dilatation of the conus carries the orifice of the pulmonary artery upwards, so that the valve comes to lie vertically over the bifurcation of the vessel, which is a fixed point. The shortening of the artery, produced by this means, renders its walls less taut, and thereby conduces to a bagging of the vessel with each systolic incursion of blood. This condition would result in the formation of eddies, and hence of a murmur.
3. The want of space necessary to accommodate the increased size of the pulmonary artery would lead to the compression of the lax arterial wall by the comparatively rigid parietes of the chest and also by the aortic arch, and this, acting as a partial constriction, would tend to the production of eddies and a murmur.
4. Owing to the greater extensibility of the anterior surface of the conus and the comparatively firm attachment of the posterior wall of the pulmonary artery to the aorta, the anterior wall of the pulmonary artery is carried up further than the posterior wall. As a consequence of this the plane of the valve ring is not perpendicular to the longitudinal axis of the artery, and the systolic incursion of blood would therefore tend to be directed against the wall of the vessel, and in this way eddies might be formed and consequently a murmur.

Dr. Foxwell also brings forward direct experimental evidence in support of his contention. Indeed, in the face of the arguments and experiments advanced by him, it is difficult if not impossible to resist the conclusion that the pulmonary systolic murmur heard in cases of cardiac debility is due to the dilatation of the conus arteriosus and pulmonary artery.

Mitral orifice.—The systolic murmur, which is audible at the apex of the heart in some cases of anæmia, is due to regurgitation through the mitral opening. It is heard most plainly in the mitral area, and is conducted with a variable degree of distinctness into the axilla, and is occasionally audible at the angle of the left scapula.

Regurgitation through the mitral orifice consequent on anæmia may be the result of—

1. Enfeeblement of the muscular fibres surrounding the opening, whereby the orifice is not sufficiently constricted to permit the proper adaptation of the valvular curtains.
2. Dilatation of the left ventricle from failure of its muscular walls, which may be due to malnutrition of the myocardium,

or to the high arterial tension that is sometimes associated with anæmia.

3. Enfeeblement of the muscoli papillares from similar causes.

Under normal conditions the closure, or in other words the proper adaptation of the curtains of the auriculo-ventricular valves, (*i.e.* mitral and tricuspid) is effected, during systole, by means of (*a*) the constriction of the orifices by the contraction of their respective muscular sphincters and (*b*) the maintenance of the levels of the valvular segments, and thus the prevention of their retroversion into the auricles by the shortening of the muscoli papillares.

Failure in the proper performance of either of these functions may prevent the perfect apposition of the valvular flaps, and thus render the valve incompetent.

Dilatation of the ventricles acts by increasing the size of the auriculo-ventricular openings, so that the contraction of the muscular sphincters cannot reduce their dimensions sufficiently to enable the curtains of the valves to come into complete apposition, and hence leakage takes place into the auricles.

Tricuspid orifice.—A systolic murmur, indicative of tricuspid regurgitation and audible in the tricuspid area, is sometimes observed with anæmia. The mechanism of its production has already been explained.

It is certainly not a little remarkable that, so far as the auscultatory evidence is concerned, mitral regurgitation always precedes leakage through the tricuspid valve in anæmia, since *a priori* it would seem likely that the right ventricle, and with it the tricuspid opening, should undergo dilatation before the left. At the same time, incompetence of the tricuspid valve, more especially when this is slight, may exist without giving rise to any auscultatory signs, and it may be that herein lies the explanation of the late appearance of the murmur.

Aortic orifice.—The systolic murmur, which may be audible in the aortic area in cases of anæmia, is in all probability due to relative stenosis of the aortic orifice, by reason of the dilatation of the left ventricle while the size of the opening remains unchanged.

Speaking generally, the area of audibility of hæmic murmurs formed at any of the cardiac orifices corresponds with that of organic murmurs, of like rhythm, produced in the same situations, though the former are seldom so widely or so clearly conducted as the latter.

Character of Hæmic Murmurs

Hæmic murmurs have usually a soft and blowing character, though those produced at the pulmonic orifice are sometimes remarkably rough and loud.

Rhythm of Hæmic Murmurs

Cardiac murmurs due to anæmia are invariably systolic as regards their time relation to the cycle of the heart.

(b) Exocardial Adventitious Sounds

1. **Friction sounds.** *Pericardial.*—In health the movements of the parietal and visceral layers of the pericardium on each other give rise to no appreciable sound. When these normally smooth surfaces become roughened by disease, friction sounds coincident with the movements of the heart may be produced. Friction sound may be audible over any portion of the præcordial area, but as a rule it appears earliest and is most distinct over the right ventricle and base of the heart. Unless it is very loud, friction sound can be heard over the site of production only, and consequently its area of audibility is usually limited by the præcordial outline; a point of some value in the differential diagnosis of pericardial from endocardial sounds, since the latter are audible outside this area.

A peculiar form of friction sound is sometimes heard over the right ventricle at the level of the fifth and sixth intercostal spaces close to the left sternal edge and over the base of the ensiform cartilage. It is possibly associated with the formation of the "white patch" which may be observed on the underlying portion of the pericardium covering the heart. The sound is systolic as regards its relation to the cardiac cycle, and may, at one time, roughly resemble a reduplication of the first sound, at another, an endocardial bruit. It may vary in character between a faint click and a definite rub, and as a rule the intensity of the sound is greatly modified by changes in the position of the body. The sound is most commonly observed in downward displacement of the heart due to emphysema, but it may be present under apparently normal conditions.

Pericardial friction sound is described as scraping, rubbing, creaking, grating, rasping, etc., and it conveys to the observer the impression that it is produced close to the ear. The intensity of the sound is comparatively uniform from beginning to end, thus differing from endocardial murmurs which usually have a crescendo or diminuendo character. Moreover, the loudness of a pericardial friction sound is commonly markedly modified by (*a*) pressure with the stethoscope, (*b*) deep inspiration or expiration, and (*c*) changes in position of the body. Furthermore, friction sound may change its site of maximum intensity from day to day—indeed it may do so while under observation—though, unlike reduplication of the heart sounds, it does not show intermissions.

Friction sound, as has already been stated, corresponds, not with the sounds of the heart, but with the movements of the organ. Consequently it usually has a "to and fro" rhythm, *i.e.*

systolic and diastolic, corresponding with the contraction and relaxation of the ventricles. It may, however, exhibit a triple rhythm, owing to the systole of the auricles, and the sound thus produced is presystolic in its relation to the cardiac cycle. The presystolic friction sound is heard most distinctly in the second and third left intercostal spaces about one and a half to two inches from the sternal border. The relation of the friction sound to the cardiac cycle may vary from day to day. At one time it may be systolic and diastolic, at another presystolic, systolic, and diastolic, or again it may be systolic or diastolic only. A sound which is observed to vary thus in its relation to the cardiac cycle is almost certainly pericardial in point of the site of its production.

It sometimes happens that both air and fluid are present at the same time in the pericardial sac. Under such conditions sounds corresponding with the movements of the heart, and having various characters, may be heard. Thus splashing or gurgling sounds are sometimes observed, and these have been compared to the noise produced by a water-wheel in motion, and hence have been called "water-wheel sounds." On other occasions an amphoric echo of the heart sounds or of pericardial friction sound is produced. These signs do not persist many days, and are invariably of the gravest import.

Pleural.—A pleural friction sound has occasionally a "to and fro" character communicated to it by the movements of the heart. In such cases the sound, as a rule, ceases when a deep inspiration is made and the breath is held.

Pleuro-pericardial.—Inflammation of the pleura over the heart may lead to the production of sounds very like those caused by pericardial friction. The differential diagnosis between the two may be difficult, but in the case of pleuro-pericardial friction sound, the site over which it is most distinctly heard is usually along the border of the left ventricle, and not over the base of the heart, or right ventricle, and further it is, as a rule, markedly modified by the respiratory movements. Thus pleuro-pericardial friction sound often disappears at the end of expiration, or when the breath is held; whereas during inspiration, when the pleural surface is more closely applied to the pericardium, it is increased in intensity. Pericardial friction sound, on the other hand, is not annulled by holding the breath, and is loudest during expiration.

2. **Murmurs.** *Cardio-pulmonary.*—It will be most convenient to consider these under sounds of exocardial origin, though, as will be seen, they are endocardial as regards site of production in many cases. They have one feature in common, which is that they all depend in the first instance on changes outside the heart.

Cardio-pulmonary murmurs are produced in a variety of ways, which may be arranged as follows :—

1. Displacements of the heart due to disease of—

- (a) The thorax
- (b) The pleuræ
- (c) The lungs
- (d) The abdomen

2. Pressure on the heart due to disease of the left pleura.

3. Changes in the lung overlying the heart.

1. Displacements of the Heart due to Disease of

(a) *The thorax*.—In severe cases of deformity of the thorax the heart is always more or less displaced, and a systolic murmur may be heard over some portion of the altered præcordial area, and is produced most probably by the dislocation and consequent twisting of the great vessels at the base of the heart.

(b) *The pleuræ*.—A systolic bruit is occasionally heard in the pulmonary or aortic area in cases of extensive pleural effusion on either side. It is in all probability produced in the pulmonary artery or aorta, as the case may be, in consequence of the slight twisting of these vessels, which may accompany considerable displacement of the heart. The murmur disappears when the fluid has been removed and the heart has returned to its normal situation.

(c) *The lungs*.—Displacement of the heart following pleuro-pericardial adhesions and the contraction of a cavity, or cirrhosis of either lung, may give rise to a systolic bruit, which is usually most distinctly heard in the second interspace to the right or left of the sternum, according as the right or left lung is affected. The murmur is also often distinctly audible over the pulmonary cavity and sometimes over the whole of the affected side. It is as a rule most intense during inspiration. The mechanism of its production is in all probability similar to that suggested in the preceding paragraph.

(d) *The abdomen*.—A systolic murmur audible at the base or apex of the heart may be due to the displacement of the organ by a large fluid or gaseous effusion into the peritoneal cavity. Removal of the effusion is followed by disappearance of the bruit.

2. Pressure on the Heart due to Disease of the Left Pleura

A systolic murmur audible over the heart and left side of the chest sometimes follows inflammation of the left pleura. In cases of this kind the lung becomes fixed over the pericardium by pleural adhesions, and the heart is more or less pressed upon by the thickened pleura. The murmur is supposed to be due to the

sudden displacement of air in the larger bronchi, as the result of the impact of the contracting ventricle on the surrounding lung tissue, which has undergone partial consolidation. The bruit is usually systolic in its relation to the cardiac cycle, but it may be diastolic, and it is heard most distinctly during inspiration. It usually disappears when the breath is held.

3. Changes in the Lung Overlying the Heart

Pulsating crepitations or râles are sometimes heard at the apex of the heart in morbid conditions of the lung overlying the organ. They disappear when the breath is held after a deep expiration.

A systolic murmur audible at the apex may be observed when the overlapping lung is partially consolidated, and its mode of production has already been explained.

A systolic murmur audible in the pulmonic area is of fairly common occurrence in tubercular disease of the upper lobe of the left lung. The pulmonary artery is in all probability displaced or pressed upon by enlarged glands, or consolidated lung, and the local narrowing of the vessel thus produced results in the formation of a fluid vein, and hence the murmur.

The Differential Diagnosis of Endocardial and Exocardial Murmurs

The size of the various chambers composing the heart should first be ascertained in the manner described under "Physical Diagnosis," and any changes that obtain should be weighed in conjunction with the known effects of any particular valvular lesion or other morbid cardiac condition which may be indicated by the presence of a murmur.

Furthermore, the condition of the pulse and the size of the liver should be ascertained. The absence of any abnormality in both of these respects would strongly contra-indicate disease of the heart.

The site of production and direction of transmission of the murmur, together with its character and rhythm, would afford valuable and, in many instances, unmistakable evidence in favour of its endocardial or exocardial origin. It is not necessary here to repeat the points which are of diagnostic value. The lungs should be examined in all cases in which a murmur of doubtful significance is heard, and the effect of the respiratory movements and of holding the breath on the abnormal sound should be carefully observed. A murmur of equivocal import in cases of tubercular disease of the upper lobe of the left lung would favour an exocardial origin, in view of the rarity with which endocardial changes occur in phthisis. Congestion of the bases of the lungs with a systolic apical murmur would strongly suggest a valvular lesion.

The effect of changes in position on the murmur sometimes affords very valuable information. A bruit which disappears when the patient lies down is almost certainly produced outside the heart.

C. VASCULAR SOUNDS

1. **Normal sounds.**—It has already been mentioned that under normal conditions the second sound of the heart is audible in the neck over the carotid and subclavian arteries. It is conducted thither by the walls of the vessels and the contained column of blood which intervene between the heart and the site of audibility of the sound.

If the stethoscope be placed lightly over the carotid, subclavian, or even femoral arteries a dull sound or thud may be heard corresponding with the ventricular systole. It has been thought that, at all events in the case of the carotid and subclavian arteries, this thud may be the first sound of the heart conducted along the walls of the intervening vessels. The sound is, however, in all probability of local origin, and is caused by the pressure of the stethoscope, and this is still more likely in the case of the femoral arteries.

Whatever be the explanation of the foregoing phenomenon, it is found that when a certain moderate degree of pressure is made with the stethoscope over the vessels in question, the sound just referred to is replaced by a murmur synchronous with the systole of the heart. It may be remarked, however, that this disposition of events is not always easy to obtain. The murmur is most probably due to a local narrowing of the vessel caused by the pressure of the stethoscope, whereby the conditions for the formation of a fluid vein are produced. A continuous murmur, audible over the internal jugular vein at the root of the neck, is explained in a similar way, and is observed under perfectly healthy conditions.

It is said that a "to and fro" murmur, *i.e.* systolic and diastolic, may be heard over the femoral artery under normal conditions, but the statement is open to serious doubt. The question will be referred to again under the next heading.

2. **Adventitious sounds.** *Arterial.*—Systolic and diastolic murmurs produced at the aortic orifice may be conducted some distance along the great arterial trunks, and are commonly to be heard in the neck. It is said that a shrill diastolic murmur of aortic origin may be audible over the radial artery at the wrist.

In cases of aortic regurgitation a combined systolic and diastolic murmur may be heard over the femoral artery.

The method of production of the systolic portion has already been considered. The diastolic moiety depends on a similar mechanism. Thus, in consequence of the incompetence of the aortic valve, there is a general reflux of blood towards the left

ventricle during diastole. As the current of blood flows backwards past the constriction in the femoral artery, produced by the pressure of the stethoscope, a fluid vein is formed, and hence a murmur. The diastolic part of the combined murmur is shorter and fainter than the systolic portion.

Under normal conditions the presence of a diastolic murmur over the femoral artery is explained by the supposition of a high degree of diastolic pressure of the pulse.

A systolic murmur audible over the subclavian arteries above and below the clavicles, and most marked on the left side, still remains to be mentioned. The murmur is modified by the respiratory movements, and may be annulled or intensified by taking a deep breath. Its mode of production is still a matter of doubt.

A systolic bruit is occasionally heard over the arteries in the neck in cases of anæmia.

Aneurismal.—The sounds which may be heard over an aneurismal sac in the neighbourhood of the heart are very variable. Most commonly two sounds, corresponding with those of the heart, are observed. The second sound is accentuated and of a lower pitch than normal, and it may be preceded by a systolic murmur. The first sound may be absent, and a systolic bruit, followed by a second sound, may alone be audible. In other instances a systolic and diastolic murmur are heard while the sounds of the heart are absent.

The systolic murmur may be conducted from the aortic orifice, or it may be produced at the mouth of the aneurism. The diastolic murmur is probably always conducted from the aortic opening.

Venous.—A continuous murmur, the “bruit de diable,” may be heard over the internal jugular veins at the root of the neck in cases of anæmia. The bruit is, however, not peculiar to anæmia. It may occur under perfectly healthy conditions, and it has been observed in 50 per cent. of cases showing no signs of anæmia.

The murmur, which has a musical humming quality, is usually most distinct on the right side. The intensity of the murmur is increased by the upright position, by turning the head away from the side that is being auscultated, and by a deep inspiration. It is also greatly modified by the degree of pressure exercised by the stethoscope over the vessel. The intensity of the murmur is also increased during inspiration and during auricular diastole.

The murmur has been ascribed to the alteration in the calibre of the vein produced by the pressure of the stethoscope and the consequent production of a fluid vein. In many cases, however, it may be heard when the instrument is placed over the sternoclavicular joint, where no pressure on the soft parts is possible.

It is probable that the anatomical relations of the lower part of

the internal jugular veins exert an important influence on the production of the murmur.

This portion of the vessel is intimately connected with the cervical fascia, so that its calibre remains constant.

If, now, the dimensions of the vein above or below this site are diminished, or increased, respectively, the adherent portion of the vessel becomes relatively dilated, or constricted, as the case may be, and these are the conditions for the formation of a fluid vein.

According to some writers a general contraction of the veins on a diminished quantity of blood in circulation is present in anæmia, while others suppose that these structures undergo relaxation from malnutrition and loss of tone. In either event the theory advanced above will explain the presence of a murmur over the internal jugular vein.

In some instances the adherent portion of the vein is normally somewhat pouched, and it may be that an alteration in the quality of the blood would, under these conditions, be sufficient to determine the formation of a fluid vein. It must be borne in mind, too, that diminished viscosity of the blood, and lessened peripheral resistance, play an important part in the production of hæmic murmurs.

A venous hum is not present in all cases of anæmia, and the intensity of the murmur does not necessarily correspond with the degree of deterioration in the quality of the blood.

A bruit may also be heard over the longitudinal and lateral sinuses, and over the subclavian and other veins in cases of anæmia.

A continuous venous murmur, apart from anæmia, is occasionally heard on either side of the xiphoid cartilage, and has been ascribed to constriction of the inferior vena cava at its junction with the right auricle.

The murmur resembles the fitful blowing of wind through the rigging of a ship under bare poles.

In those exceedingly rare instances in which a communication is formed between the ascending aorta and superior vena cava, a murmur may be heard over the first or second right intercostal space an inch or more from the sternal edge, which bears a resemblance to the sound produced by a water-wheel in motion.

The continuous murmur which attends the establishment of a communication between the aorta and pulmonary artery, a very rare event, is heard best close to the left sternal edge, about the level of the second costal cartilage. The murmur heard under these circumstances, though continuous, is not uniform in intensity, but varies rhythmically with the balance of pressure in the two vessels.

CHAPTER IV

THE PULSE

Definition—Method of Production—Physical Examination of Pulse—Inspection
—Palpation—Graphic Record or Sphygmogram—Instrumental
Determination of the Blood Pressure.

FROM a clinical point of view the pulse is the alteration in the shape of an artery which obtains during the time that each wave of increased pressure, due to the heart's systole, passes along the vessel. The perception of the pulse rests on the visible and palpable displacement which the artery imparts to the media in contact with it, as each wave of increased pressure passes beneath the point of contact.

The pulse depends on—

1. An alteration in the shape of the artery from the flattened to the cylindrical (Broadbent).
2. A slight expansion of the artery.

An artery is usually flattened by the tissues which overlie it, and exercise pressure against some underlying and resistant medium. In the case of the radial artery it is the radius against which the vessel is pressed. The artery is still further flattened by the examining finger. All elastic tubes, however, tend to become circular when the fluid pressure within them is sufficient to overcome the resistances which conduce to alterations in their shape. Thus it is that as the pulse wave passes any particular point in the course of an artery, the vessel's shape is altered from the flattened to the circular, and a false impression of expansion is experienced. In addition to this factor in the production of the pulse the arterial wall does actually expand, but, in the case of the radial artery, to an extent which could scarcely be appreciated by the finger.

The physical examination of the pulse includes—

1. Inspection
2. Palpation
3. Graphic record or sphygmogram
4. The instrumental determination of the blood pressure.

INSPECTION.

The information derived from inspection of the pulse is always checked by subsequent palpation. Nevertheless this method of investigation has its value as a means of rapid diagnosis. The vessels which lend themselves most readily to this mode of examination are the temporal arteries, and occasionally the retinal arteries, since the course of these vessels can usually be seen in the adult by careful inspection. The degree of tortuosity of the artery, and the frequency, regularity, and to some extent the character of the pulse, as well as the bilateral symmetry of the pulsation, can be roughly gauged. Thus aortic regurgitation may be suspected in cases where the discursion of the pulse is extensive, the collapse of the pulse wave sudden, and the vessels tortuous. Heart failure, especially in mitral regurgitation, is suggested by inequalities in the force and rhythm of the visible pulse. Visible pulsation of the carotid arteries is frequently associated with aortic regurgitation, and also with exophthalmic goitre and other nervous disorders of the heart. Other points, which may be observed on inspection, have their diagnostic value as described under palpation of the pulse.

PALPATION

Method of Feeling the Pulse

The first three fingers should be placed lightly upon the radial artery at the wrist, with the forefinger nearest the heart and the thumb supporting the wrist. The vessel should be investigated, under varying degrees of pressure, both in its transverse and longitudinal aspects, and each feature of the pulse that requires attention should be appreciated by a distinct and well-defined manœuvre of the examining fingers.

The features of the pulse to which the observer's attention should be directed are the following :—

1. The frequency. While counting the pulse rate, the regularity or irregularity of the force and rhythm of the pulse should also be observed.
2. The size of the artery.
3. The degree of fulness of the artery between the pulsations.
4. The character of the pulse wave.
5. The compressibility of the vessel.
6. The condition of the arterial wall.
7. The bilateral symmetry of the pulsations.

1. **The frequency of the pulse.**—The rate of the pulse and of the heart beats usually correspond ; but this is not always the case, inasmuch as the force of the cardiac systole may not be sufficient to propel the pulse wave as far as the radial artery. The average

rate of the pulse in the adult male is about seventy-two beats per minute.

The conditions which give rise to physiological variations in the frequency of the pulse are: (1) Age, *i.e.* the pulse rate is quicker in children than in adults; (2) Sex, *i.e.* the pulse rate is quicker in women than men; (3) Heredity; (4) Nervous impressions; (5) Emotional disturbance; (6) Exertion; (7) Position; (8) Food; (9) Temperature; (10) Time of day or night; (11) Alterations in blood pressure, etc.

The pulse rate is also influenced by drugs, such as alcohol, tobacco, digitalis, and the like.

The variations in the pulse rate produced by disease may be tabulated as follow:—

A. Increased Frequency of the Pulse

- (1) Pyrexia. (2) Anæmia. (3) Pathological conditions which decrease blood pressure. (4) Pericarditis. (5) Myocarditis. (6) Valvular disease of the heart. (7) Dilatation of the heart. (8) Irritable heart (Da Costa). (9) Loss of vagus control, or irritation of the cervical sympathetic (accelerator) nerves as observed in cases of palpitation, ex-ophthalmic goitre, etc. (10) Tachycardia. (11) Hysteria.

B. Diminished Frequency of the Pulse

- (1) Renal disease. (2) Pathological conditions which increase blood pressure. (3) Jaundice. (4) Fatty and occasionally fibroid disease of the heart (the pulse rate is, however, sometimes increased under these circumstances). (5) Epilepsy and other cerebral disorders. (6) Pain. (7) During convalescence from the acute fevers, such as pneumonia, typhoid, etc.

The diminished frequency of the pulse that is found in association with the remarkable condition in which two beats of the heart occur to one of the pulse is of course not included in the present category. The condition is mentioned here with the object of emphasizing the necessity of controlling observations made with respect to the frequency of the pulse at the wrist by an examination of the heart.

Rhythm

The rhythm of the pulse and of the heart's action usually correspond, but, as in the condition just mentioned, this is not necessarily the case.

Deviations from the normal rhythm give rise either to "intermittence" or to "irregularity" of the pulse, or to a combination of these conditions.

Intermittence of the pulse means the omission of a beat, which may occur at regular or irregular intervals. The phenomenon is more commonly observed in old than in young people, and it is fre-

quently found independent of any other discoverable abnormality. It is habitual in some individuals, while in others it is readily produced by emotional disturbance, indigestion, or the abuse of tea and tobacco. Intermittence of the pulse is also observed in association with gouty manifestations. It is sometimes found in connection with fatty disease of the heart, with cardiac failure, and with acute affections of the lungs, and is then of serious import. It is said that habitual intermittence of the pulse usually disappears during attacks of pyrexia.

Irregularity of the pulse usually, but not necessarily, implies inequalities in the force and volume of the pulsations, as well as the appearance of the beats at unequal intervals of time. It is associated with valvular disease of the heart, and more especially with mitral regurgitation. It is also commonly observed in connection with failure of the heart from any cause, and speaking generally, it is a sign of disturbance of myocardial metabolism. Irregularity of the pulse sometimes depends on reflex disturbance of the heart from gastro-intestinal and uterine disorders. It also occurs in association with the abuse of tea, coffee, and tobacco. It is occasionally found apart from any other morbid manifestations.

Certain peculiar modifications in the rhythm of the pulse are designated under the special titles of the *pulsus bigeminus*, the *pulsus trigeminus*, the *pulsus alternans*, and the *pulsus paradoxus*.

Pulsus bigeminus.—In this variety of pulse the beats are grouped in pairs, with a pause between each group. The second beat is usually the weaker of the two. The heart beats correspond in rhythm with the pulse, so that a strong impulse is followed by a weak one. This variety of pulse is found most commonly in mitral stenosis, more especially when under the influence of digitalis. It is also observed in association with bodily and mental strain and with epileptiform attacks.

Pulsus trigeminus.—In this variety of pulse the beats are arranged in groups of three. It occurs under conditions similar to those in which the *pulsus bigeminus* is observed.

Pulsus alternans.—The regular succession of a strong and weak pulsation constitutes the *pulsus alternans*, which is observed in connection with Cheyne-Stokes' respiration, (Sanson) and with the other conditions of central nervous disturbance, and occasionally also with mitral affections.

Pulsus paradoxus.—In this condition the pulse is markedly influenced by the respiratory movements. During inspiration the pulse wave is annulled, or becomes much diminished in force, while during expiration it may be of full amplitude. It can sometimes be elicited under physiological conditions by holding the breath in extreme inspiration or expiration. In certain pathological conditions, however, it is more or less constantly present. It occurs in association with pericardial adhesions, mitral stenosis, emphysema, and with conditions of heart failure.

So far as the mechanism of production of this variety of pulse is concerned, it is probable that the increase of negative pressure in the thorax with each inspiration offers a greater resistance to the discharge of the ventricular contents than an enfeebled heart is able to cope with.

The pulsus paradoxus is of small diagnostic value.

2. The size of the artery.—Observations on this head are of importance, inasmuch as the size of the artery appreciably affects the perception of the size and force of the pulse wave. Thus the pulse wave in a large artery will appear more forcible than in a small one, but, on the other hand, the pulsation is much more easily obliterated by pressure with the fingers in the former case than in the latter. The size of the artery, therefore, becomes one of the factors in the estimation of the strength or force of the pulse.

The size of the artery depends for the most part on the condition of contraction or relaxation of the vessel, which is of course regulated by the tone of the arterial walls. The thickness of the arterial wall also effects to a slight extent the size of the vessel.

The size (calibre) of the artery is most accurately determined by means of Oliver's arteriometer.

The systematic use of this instrument is of very great service, inasmuch as it enables a record to be kept of the calibre of the artery for purposes of comparison and reference.

3. The degree of fulness of the artery between the pulsations.—The degree of fulness of the artery between the beats of the pulse is the measure of the mean intra-arterial pressure, or pulse tension, and is determined by (1) the force of the ventricular systole, (2) the peripheral resistance, and (3) the competency of the aortic valve.

In order to determine the tension of the pulse the fingers should be carried transversely to and fro across the artery, and an attempt made to roll the vessel beneath the fingers.

When the artery can be felt distinctly during the passage of the pulse wave beneath the finger, and is, with care, distinguishable between the pulsations, the pulse may be regarded to be of average tension. When the artery cannot be clearly outlined by the fingers at any period of the passage of the pulse wave, or during the interval between the pulsations, the pulse is said to exhibit low tension. When the artery can be rolled beneath the fingers during and between the pulsations the pulse is described as one of high tension.

An artery that is empty between the beats of the pulse (low-tension pulse) depends on a general want of tone of the ventricular and arterial walls, and is occasioned by—

- (1) Pyrexia. (2) Hereditary influences. (3) General debility, due to anxiety or nervous strain of any kind, excesses of various kinds, bad hygienic surroundings, anæmia, and so forth.
- (4) Food. (5) Serious purgation. (6) Exhaustion or fatigue from any cause. (7) Fatty degeneration of the heart. (8) Dilatation of the heart. (9) Aortic regurgitation. (10) Mitral regurgitation, etc.

An artery that is full between the beats of the pulse (high-tension pulse) depends on (1) increase in the quantity of blood in the circulation, (2) forcible action of the heart, (3) increased arterial tone and arterio-capillary resistance, and is occasioned by—

- (1) Age. (2) Hereditary influences. (3) Renal disease. (4) Gout. (5) Pregnancy. (6) Lead poisoning. (7) Anæmia (in some cases). (8) Diabetes (in some cases). (9) An attack of angina pectoris. (10) Constipation. (11) Peritonitis. (12) Emphysema and chronic bronchitis. (13) Aortic and mitral stenosis, etc.

4. **The character of the pulse wave.**—The character of the pulse wave must be considered with regard to its rise, duration, and fall. The rise of the pulse wave may be sudden or gradual, of great or of small amplitude.

It is sudden and of great amplitude when a comparatively large amount of blood is pumped into an empty arterial system, as in (1) aortic regurgitation with hypertrophy of the heart, (2) after hæmorrhage, (3) in most pulses of low tension. The rise of the pulse wave is gradual if the ventricle can empty itself slowly only into the aorta, as in (1) aortic stenosis, (2) aneurism of the aorta, (3) high-tension pulses.

The rise of the pulse wave is of small amplitude (1) when the pulse tension is high, (2) when the amount of blood ejected into the aorta is small, as in mitral stenosis, mitral regurgitation, etc.

The duration of the pulse wave is considerable when the pulse tension is high and the ventricular systole powerful, as in cases of high tension, aortic stenosis, etc.

The duration of the pulse wave is short when the pulse tension is low or the ventricular systole is weak, as in low-tension pulses and failure of the heart, etc.

The fall of the pulse wave may be sudden or gradual, with or without subsidiary pulsations. A sudden fall of the pulse wave is observed in cases of (1) aortic regurgitation, (2) pulses of low tension.

A gradual fall of the pulse wave is observed in (1) aortic stenosis, (2) pulses of high tension, (3) aneurism.

Secondary pulsations, etc.—The dicrotic wave is practically the only secondary or subsidiary pulsation appreciable to the finger. It is most commonly observed when the pulse tension is low and the heart beat forcible. A fuller account of the dicrotic pulse and of the other subsidiary waves that may be observed will be given under the account of the sphygmogram.

5. **The compressibility of the artery.**—The strength of the pulse, and the degree of the constant or mean intra-arterial pressure or tension, is computed from the compressibility of the artery. The degree of pressure that is required to obliterate the pulse is the measure of its strength. The degree of intra-arterial pressure is

gauged by the amount of force that is required to flatten the artery between the pulsations.

The method of estimating the compressibility of the artery and hence of the variable and constant pressure within the vessel is as follows: Three fingers are placed upon the radial artery in the manner previously described. The two fingers nearest the heart exercise pressure upon the artery until the pulsations are obliterated, that is, they cannot be felt by the third finger. The degree of pressure that is required to extinguish the pulse is the measure of its strength. The three fingers may be used to compress the vessel between the beats, and the degree of pressure that is required to flatten the artery is the guide to the estimation of the mean arterial tension.

The artery is difficult to compress in (1) pulses of high tension, (2) when the vessel is rigid from degenerative changes in its walls.

The artery is easy to compress in pulses of low tension.

6. The condition of the arterial wall.—In order to estimate the condition of the arterial wall, the fingers should carry the skin up and down along the course of the vessel with varying degrees of pressure.

The healthy arterial wall is soft and elastic to the touch. On passing the fingers along the course of the artery and applying some pressure, no variations in consistency, or irregularity in outline should be felt. If the vessel feels unnaturally rigid, with irregularities in thickness and density, especially when this is combined with tortuosity of outline, the arterial wall has undergone degeneration.

Hardened patches are due to established or commencing calcareous deposition, and the tortuosities depend on the loss of elasticity of the arterial coat in conjunction with strain on the vessel. In extreme cases the artery is found to be converted into a rigid calcareous tube.

Atheroma is a frequent sequel of protracted high arterial tension. It is usually present to a greater or less degree in old age.

7. The bilateral symmetry of the pulsations.—In cases of doubtful nature it is well to make a practice of examining the pulse at each wrist simultaneously, with the object of disclosing any want of synchronism, or differences in the size or character of the two pulses that may obtain.

Apart from an abnormal distribution of the arteries, a want of synchronism, or a marked difference in the size or character of the two radial pulses, is usually indicative of aneurism of one or other of the great arterial trunks.

Recurrent pulse.

This pulse, which is felt as a feeble and retarded beat on the distal side of the radial artery after this vessel has been completely blocked by pressure with the fingers, is due to the transmission of a

wave through the palmar arch by way of the ulnar artery. It is observed in cases of extremely low tension, with a vigorous ventricular contraction, as, for instance, in aortic regurgitation, and also in conditions where protracted high tension has led to failure of the muscular coats of the arteries, as, for example, may obtain in the terminal stages of chronic Bright's disease.

GRAPHIC RECORD OR SPHYGMOGRAM

For a description of the sphygmograph and its method of use the reader is referred to works on physiology.

The sphygmogram is a tracing of the discursions of the pulse magnified by the lever mechanism of the sphygmograph, and registered on a recording surface. It consists of—

1. An ascending limb (*a-b*)
2. An apex (*b*)
3. A descending limb (*b-g*), which is interrupted by one or more secondary waves and notches, viz.—
 - (*a*) The tidal or predicrotic wave (*d*)
 - (*b*) The aortic notch (*e*)
 - (*c*) The dicrotic wave (*f*)
 - (*d*) Subsidiary or post-dicrotic waves

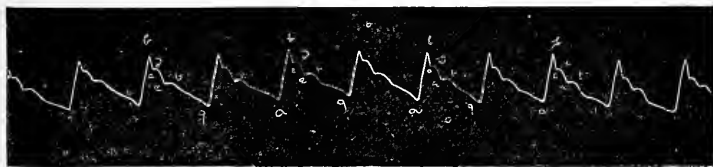


FIG. 14. NORMAL PULSE TRACING

1. The Ascending Limb

In health this line is vertical, or nearly so. Its amplitude depends on—

1. The energy of the heart's beat
2. The intra-arterial tension
3. The magnifying power of the lever mechanism
4. The skill of the operator

The energy of the heart's beat.—*Ceteris paribus*, the more energetic the beat of the heart the greater the amplitude of the

ascending limb of the pulse trace, but owing to the correlation between the cardiac and vascular mechanisms, it usually happens that when the heart's beat is forcible the arterial tension is high. Hence the effect of a powerful ventricular contraction on the amplitude of the ascending limb of the sphygmogram is largely neutralized.

The intra-arterial tension.—When the arterial tension is high the artery is circular, not only during the passage of the pulse wave, but also during the interval between the pulsations, unless the vessel be subjected to great pressure. Hence the full effect of the change of shape in the artery, *i.e.* from the flattened to the circular, loses its due influence on the percussion stroke, which, in consequence, is of low amplitude. When, on the other hand, the tension is low the artery is flattened between the beats of the pulse by the pressure of the small arm of the lever of the sphygmograph; and hence the discursion of the long arm of the lever, when the vessel becomes circular, is considerable or great as the case may be.

The skill of the operator.—Unless the lever be accurately adjusted over the artery, the displacement of the tissues surrounding the vessel, and not that of its walls, is imparted to the sphygmograph. There is for every degree of tension a degree of pressure which produces the maximum discursion of the lever. This can be found by experiment only, and by the skilled operator.

The ascending limb of the sphygmogram is of great amplitude in—

1. Aortic regurgitation
2. Pulses of low tension with forcible heart beat
3. The pulse after hæmorrhage

The ascending limb is of small amplitude in—

1. Pulses of high tension
2. Mitral disease
3. Aortic stenosis
4. Aneurism

2. The Apex (*δ*)

The apex is the angle included between the ascending and descending limbs of the sphygmogram. It is normally acute. Its arc, however, largely depends on the rate of movement of the recording surface.

The angle is very acute under those conditions which produce an ascending limb of great amplitude. It is obtuse when the arterial tension is high, or when the aorta is slowly filled, as in aortic stenosis, and aneurism of the arch of the aorta. Sometimes the tracing has a double apex (*pulsus bisferiens*)—a condition which sometimes obtains in aortic stenosis and in cases of senile degenera-

tion of the arteries. It is compounded of two distinct efforts on the part of the ventricle to empty itself.

3. The Descending Limb (*b-g*)

The first part of this line (*b-e*) is due to the fall of the lever after its initial elevation by the pulse wave. Together with the ascending limb and the apex it forms the so-called percussion wave.

Tidal Wave (*D*)

The explanation of the cause and significance of the tidal wave is most succinctly and clearly given by Mahomed in the following words :—

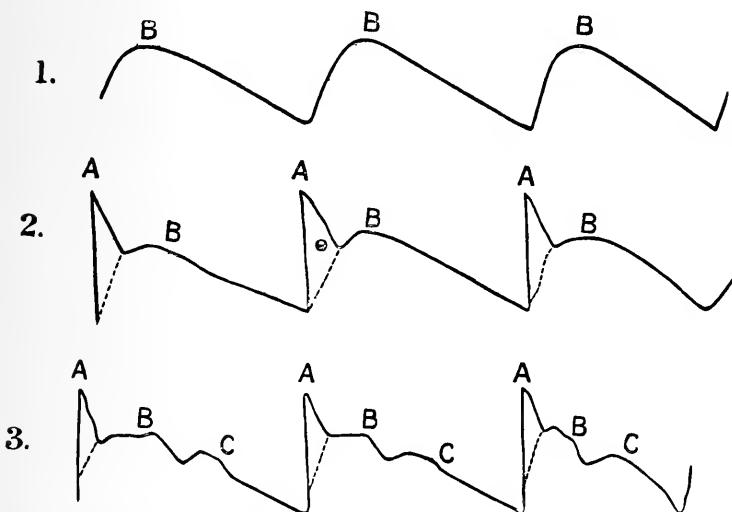


FIG. 15. MAHOMED'S SCHEME OF THE TIDAL WAVE

"The simplest pulsatile movement that can be conceived in an elastic tube is the mere passage of a wave of fluid through it, causing more or less sudden expansion and a gradual collapse of the tube as it passes through it; such a wave is the foundation of the pulse, and has been called the 'Tidal' wave. If the impulse imparted to the fluid is more sudden, an element of percussion or shock will be introduced (Fig. 2), giving an abrupt and vertical up-stroke from the jerking of the lever by the sudden expansion of the artery [change of shape.—Author]. Owing to its acquired velocity, this movement of the lever is rather greater than the corresponding movement in the arterial wall which produced it, and on reaching its highest point, it falls suddenly by its own weight, till it

is again caught and perhaps slightly raised by the tidal wave *B*, which is now only reaching its maximum of distension.

" . . . The tidal wave is the true pulse wave, and indicates the passage of a volume of blood through the arteries, pumped into them by each contraction of the heart. It resembles the passage of the tidal wave or 'bore' up a river; hence its name. It is transmitted more slowly than the percussion wave, or rather attains its maximum intensity more gradually; hence their separation in the tracing. Though they usually commence to distend the artery together, the percussion wave necessarily attains its maximum intensity instantaneously, it being only a shock, while the tidal wave does so more gradually. Sometimes a considerable interval elapses between them. Frequently they are inseparable, the percussion wave not existing or else being merged into the tidal."

The tidal wave has been explained on the assumption that it is a reflection of the primary wave from the periphery, but the view advanced above is perhaps the one most commonly held.

Aortic Notch and Dicrotic Wave (*e* and *f*)

The recoil of the elastic arterial walls, which follows their distension by the percussion and tidal waves, forces the blood backwards and forwards towards the aortic cusps and towards the periphery. The flow backwards closes the aortic valve (represented by the aortic notch), and the barrier thus thrown in the path of the blood stream gives rise to a reflected wave of increased pressure (the dicrotic wave). Other views are held with regard to the causation of the dicrotic wave, but the explanation given here is the one most commonly accepted.

The subsidiary waves which sometimes appear on the descending limb of the sphygmogram below the dicrotic wave are echoes of the dicrotic wave. They are due to the vibrations set up in the elastic arterial walls consequent on the injection of the mass of blood into the aorta at each systole of the heart. As equilibrium is established they gradually die away. Irregular undulations on the descending limb of the pulse trace are sometimes due to vibrations of the sphygmographic lever, or of the tissues in the immediate neighbourhood of the pulse.

The application of the foregoing methods of examination of the pulse will now be illustrated in the following brief description of the characteristic features of the pulse in—

1. High tension
2. Low tension
3. Virtual tension
4. Hypertrophy of the heart
5. Dilatation of the heart
6. Aortic regurgitation
7. Aortic stenosis

8. Combined aortic regurgitation and stenosis
9. Mitral regurgitation
10. Mitral stenosis
11. Combined mitral regurgitation and stenosis
12. Combined aortic and mitral disease
13. Aneurism of the arch of the aorta

1. High-tension Pulse

Inspection.—Unless its walls are degenerated the vessel is usually small and contracted. If degenerated, the temporal arteries stand out as thick sinuous cords. The radial artery, under these circumstances, is visible far up the arm, and the vessel is thrown into curves with each pulsation. The diameter of the artery does not appear to be appreciably altered during the passage of the pulse wave.

Palpation.—The pulse is decreased in frequency, but regular in force and rhythm. The vessel is small if healthy; large if degenerated, full between the beats and not easily compressible. The pulse wave is of low amplitude, rises slowly, lasts long, falls slowly,

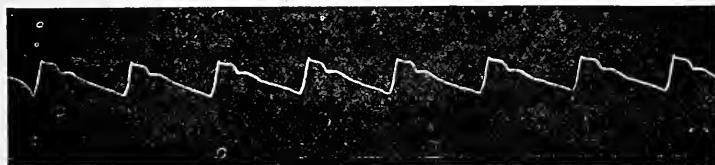


FIG. 16. HIGH-TENSION PULSE TRACING

and is difficult to compress. The arterial wall, usually more or less thickened, is sometimes extremely degenerated.

Sphygmogram.—The ascending limb is of low amplitude, and shows a gradual, somewhat slanting ascent. The percussion wave is badly defined and often absent. The apex (tidal wave) is rounded. The dirotic wave is small, and situated high up on the descending limb. The descending limb falls gradually, and is interrupted by numerous subsidiary waves.

2. Low-tension Pulse

Inspection.—The pulse is often visible as an energetic throb. The vessel cannot be seen between the beats.

Palpation.—The pulse is increased in frequency, but regular in force and rhythm. The artery, which may be large or small, cannot

be felt between the beats. The pulse wave, which may be of great or small amplitude, in accordance with the force of the ventricular contraction, rises quickly, is of brief duration, falls quickly, and is easily obliterated by pressure.

Dicrotism is always well marked, so that in some instances two distinct pulsations can be felt with each beat of the heart. This variety of low-tension pulse has been termed the dicrotic pulse.

The arterial wall is healthy.

Sphygmogram.—The ascending limb rises quickly, and is of great or low amplitude according as the left ventricle is acting powerfully or feebly. The apex is acute and the tidal wave badly marked.



FIG. 17. LOW-TENSION PULSE TRACING

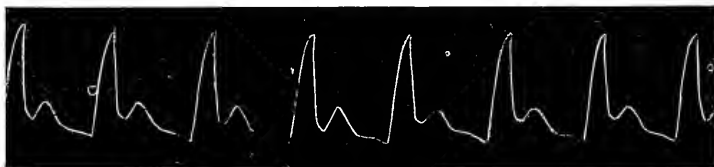


FIG. 18. DICROTIC PULSE TRACING

The aortic notch and dicrotic wave are well marked and are situated low on the descending limb, which falls suddenly.

3. Virtual-tension Pulse

This variety of pulse is observed when the element of forcible and energetic heart beat is wanting, but the other conditions are those productive of high tension. These conditions obtain in the terminal stages of Bright's disease, when the heart is failing, and in mitral stenosis, when the amount of blood discharged at each systole into the aorta is small.

Inspection.—If the pulse is visible (*i.e.* in the last stages of Bright's disease) it rises and falls suddenly. The artery is large and its walls tortuous.

Palpation.—The pulse may or may not be increased in frequency. It is usually regular in force and rhythm until the heart fails (mitral stenosis). The vessel is usually large, but it may be small, as in mitral stenosis.

The artery is full between the beats and fairly easily compressible. The wave rises rather suddenly, is of short duration, and falls somewhat less abruptly than it ascends. The wave is easily compressed, and the vessel wall is usually more or less degenerated.

Sphygmogram.—The ascending limb is steep and of considerable amplitude, the apex is acute and the tidal wave short. The descending limb is somewhat steep and the dicrotic wave is fairly well marked, and is situated well above the base line.

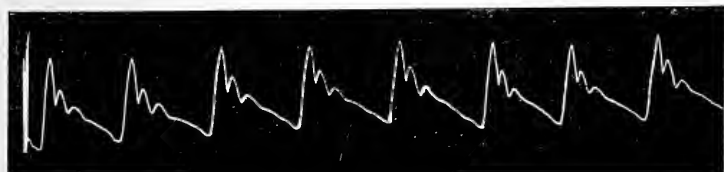


FIG. 19. VIRTUAL-TENSION PULSE TRACING

4. Hypertrophy of the Heart

The pulse in cases of cardiac hypertrophy is that of the condition which has given rise to the enlargement of the organ. Simple hypertrophy of the heart from prolonged over-exertion is associated with the following pulse :—

Inspection.—The arteries are often large, and show regular and forcible pulsation.

Palpation.—The pulse is normal in frequency and regular in force and rhythm. The vessel is often large, and can usually be felt between the beats. The wave rises suddenly, falls rather rapidly, and is fairly easily compressible. The arterial wall is healthy.

Sphygmogram.—Normal, but on rather a large scale.

5. Dilatation of the Heart

As in the case of hypertrophy, dilatation of the heart is usually associated with conditions which modify the characters of the pulse.

The pulse of dilatation of the heart due to over-exertion, or to weakness of the cardiac walls following pyrexia, the acute fevers, etc., needs a brief description.

Inspection.—Pulse not visible.

Palpation.—The frequency of the pulse is increased. The pulsations are irregular in force and rhythm. The vessel is small and cannot be felt between the beats. The wave is small, and it rises and falls quickly. The arterial wall may or may not be degenerated.



FIG. 20. PULSE TRACING FROM A CASE OF DILATATION OF THE HEART SHOWING RESPIRATORY CURVES

Sphygmogram.—The tracing is very irregular. The dicrotic wave is usually present.

6. Aortic regurgitation

The collapsing pulse of Corrigan, or the water-hammer pulse.

1. In valvular disease
2. In atheroma

1. IN VALVULAR DISEASE

Inspection.—The arteries of the neck, the temporal, subclavian, brachial, and radial arteries, etc., show visible and often violent pulsation, which exhibits a rapid rise and fall. The arteries are usually tortuous and more or less thickened and degenerated. The tortuosity is increased and is associated with a peculiar vermiform movement of the vessel as the pulse wave passes along it.

Palpation.—The pulse is normal in frequency and regular in force and rhythm. The vessel is large and empty between the beats, a feature that is more readily appreciated by raising the wrist above the level of the shoulder. The wave rises very suddenly and falls with remarkable abruptness, and is of momentary duration only. It is easily compressible. The arterial wall is usually thickened and may be degenerated.

It will be noted that there is an appreciable delay in the appearance of the pulse wave at the wrist after the systole of the heart. This loss of time between the systole of the ventricle and the appearance of the pulse wave is peculiar to low-tension pulses, and reaches its maximum development in cases of aortic incompetence.

Capillary and venous pulsation are other phenomena which may be seen in connection with a relaxed condition of the arterial system, such as obtains in cases of aortic regurgitation. In these conditions the relaxed arterial walls fail to obliterate the pulse wave, which is therefore carried on into the capillaries and thence into the veins.

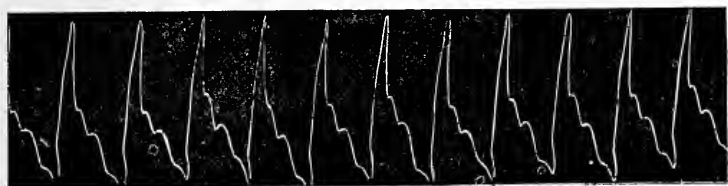


FIG. 21. PULSE TRACING FROM A CASE OF AORTIC REGURGITATION

Sphygmogram.—The ascending limb is steep and of great amplitude. The apex is very acute, and the descending limb remarkably steep. The tidal and dicrotic waves are hardly marked on the tracing, or are altogether wanting.

2. AORTIC REGURGITATION DUE TO ATHEROMA

The regurgitation in these cases is due for the most part to dilatation of the root of the aorta involving the aortic orifice. The degree of regurgitation is commensurate with the degree of dilatation of the orifice and is usually slight.

Inspection.—The temporal and radial arteries may stand out prominently as tortuous cords. The pulsation, when visible, will appear to come and go quickly, and may be attended by locomotion of the arteries.

Palpation.—The pulse is normal in frequency and regular in force and rhythm. The artery is large and full between the beats. The wave rises and falls suddenly, and is easily compressible. The wall of the artery will show degenerative changes. The delay of the pulse at the wrist is very much less marked than in aortic regurgitation due to valvular disease.

Sphygmogram.—The ascending limb is of moderate amplitude and somewhat steep. The descending limb is moderately steep and shows an ill-marked dicrotic wave.

7. Aortic Stenosis

Inspection.—The pulse is not usually visible.

Palpation.—The pulse is normal in frequency and regular in force and rhythm. The artery is small and can be felt between the beats.

The pulse wave rises slowly, falls slowly, and is of considerable duration. It is fairly easily compressible. The condition of the arterial wall is normal.



FIG. 22. PULSE TRACING FROM A CASE OF AORTIC STENOSIS

Sphygmogram.—Percussion wave absent. The tidal wave has an ascending limb of low amplitude, rising gradually. The apex is rounded or flattened. The descending limb falls gradually. The dirotic wave is badly marked.

In cases of aortic stenosis, and also in other forms of cardio-arterial disease, tracings with a double summit are sometimes

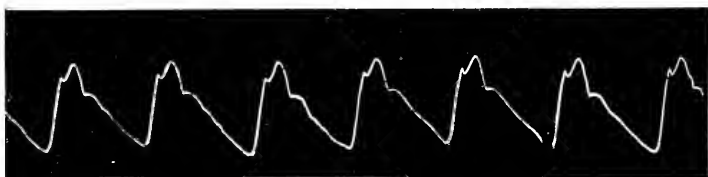


FIG. 23. ANACROTIC PULSE TRACING



FIG. 24. PULSUS BISFERIENS

obtained. This is probably due to an ineffectual percussion wave followed by a tidal wave of greater amplitude. According to Broadbent, the double summit is an example of the pulsus bisferiens, and is due to a reinforcement of the ventricular systole towards its close.

8. Combined Aortic Regurgitation and Stenosis

The pulse is the summation of the effects produced by the two lesions, with the result that the characters proper to each disease are largely neutralized.

Speaking generally, the pulse will reflect, for the most part, the characters peculiar to the predominating lesion.

As a rule the pulse is visible, regular, and more or less collapsing.

9. Mitral Regurgitation

Inspection.—The pulse is not usually visible.

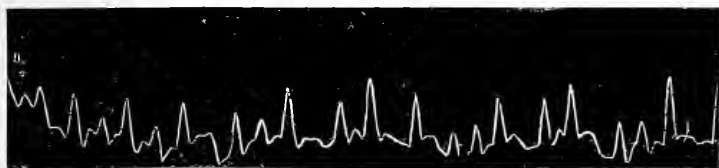


FIG. 25. PULSE TRACING FROM A CASE OF MITRAL REGURGITATION

Palpation.—The pulse is increased in frequency and is irregular in force and rhythm. The artery is small and cannot be felt between the beats. The wave rises and falls quickly, and is easily compressible. The arterial wall is healthy.

Sphygmogram.—The tracing is often very irregular. The ascending limb is of low and irregular amplitude. It rises rapidly. The apex is usually sharp, and the ascending limb falls quickly. The dicrotic wave is fairly well marked, and usually appears low down on the descending limb.

10. Mitral Stenosis

Inspection.—The pulse is not visible.

Palpation.—The pulse is slightly increased in frequency, and is regular in force and rhythm until the heart fails. The artery is small and full between the beats. The wave is small and rises and falls gradually. It is easily compressed. The arterial wall is healthy.



FIG. 26. PULSE TRACING FROM A CASE OF MITRAL STENOSIS

Sphygmogram.—The ascending limb rises gradually and is of small amplitude. The apex is usually rounded. The descending limb falls gradually and shows a badly marked aortic notch and dicrotic wave.

In the later stages of mitral stenosis, with the establishment of tricuspid regurgitation, etc., the pulse becomes very irregular, owing partly to the fact that many abortive heart beats fail to propel the pulse wave as far as the wrist.

11. Combined Mitral Stenosis and Regurgitation

The pulse is very irregular and small. It combines the features peculiar to the two lesions, the influence of the predominant affection preponderating.

12. Combined Aortic and Mitral Disease

The pulse differs greatly in different cases. The predominating lesion exercises the greater influence on the characters of the pulse.

If aortic regurgitation predominates, the percussion wave is large and collapse of the pulse wave prominent. If mitral regurgitation, irregularity is the main feature. If aortic stenosis, the percussion wave is absent, the tidal wave taking its place. If mitral stenosis, the smallness of the pulse is its most distinguishing feature.

13. Pulse of Aneurism

The radial pulses on the two sides may differ in time and character.

Inspection.—A visible pulsation may occur over the seat of the aneurism.

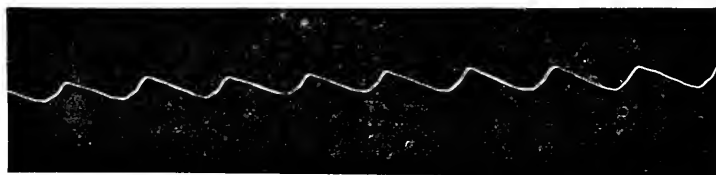


FIG. 27. PULSE TRACING TAKEN FROM THE LEFT RADIAL ARTERY IN A CASE OF ANEURISM

Palpation.—The pulse is normal in frequency and regular in force and rhythm. The artery is small and full between the beats. The wave is small, gradual, prolonged, and subsides gradually. It is easily compressible.

Simultaneous examination of the two radial arteries may show delay and altered character of the pulse on the affected side.

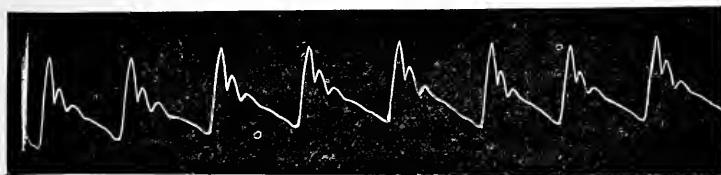


FIG. 28. PULSE TRACING TAKEN FROM THE RIGHT RADIAL ARTERY
IN THE SAME CASE (FIG. 27)

Sphygmogram.—The percussion wave is absent. The ascending limb of the tidal wave is of low amplitude and rises slowly. The apex is rounded, and the descending limb falls gradually. The dicrotic and secondary waves are usually absent (Fig. 27).

THE INSTRUMENTAL DETERMINATION OF THE BLOOD PRESSURE

The digital estimation of the blood pressure obtained from a peripheral vessel like the radial artery is liable to several fallacies which may mislead even the well-trained finger (Oliver). In order to ensure accuracy of observation, and also to provide a record for the purposes of comparison and reference, it is necessary to employ other means of gauging the blood pressure. Instruments which fulfil these requirements have been devised by Hill and Barnard (the Sphygmometer); also by Oliver (the Hæmodynamometer), and by others. The estimation of the blood pressure by both instruments is obtained through a fluid medium, and herein lies the accuracy of the method.

The sensitiveness and accuracy of these instruments have been thoroughly tested, and though the results obtained are not beyond dispute, it is not too much to say that a blood pressure gauge should form part of the equipment of every clinician. Both the arterial and venous blood pressures are capable of measurement. Oliver states that if the influence of gravitation be excluded the arterial blood pressure is practically uniform throughout the arterial system. The average mean arterial pressure varies between 90 and 110 c.mm. Hg. It is modified by the age, weight, and build of the individual. The average venous pressure in recumbency Oliver puts at 10-20 c.mm. Hg.

CHAPTER V

THE CARDIOGRAPH

Its Sphere of Usefulness—The Normal Cardiogram—Cardiograms of the Chief Valvular Lesions.

THE value of the cardiograph as a means of diagnosis even in skilful hands is somewhat problematical, and in any case the results obtained are decidedly less trustworthy than those afforded by the sphygmograph. The apex beat is often difficult or impossible to define, and unless the button of the instrument is accurately applied over the site of its manifestation, the tracing obtained is not that which is due to the actual impulse of the heart against the chest wall, but to movements in the neighbourhood of the apex beat which give the so-called "inverted tracing." If the operator is not aware of this possibility his results may be quite unintelligible.

Provided, however, that an accurate and reliable tracing can be



FIG. 29. NORMAL CARDIOGRAM
(Sansom after Galabin)

- F = Closure of semilunar valves and commencement of diastole
- K = Elevation due to sudden filling of ventricle by the active dilatation of its walls
- l = Period of passive filling of ventricle
- a = Auricular systole and completion of the process of filling the ventricle
- a-d = Sudden rise of lever due to contraction of the ventricular wall at the commencement of systole
- d = Sudden opening of aortic valve
- d e f = Continued contraction of ventricular walls
- f = Closure of aortic valve

Note.—a-f=systole
f-a=diastole

obtained, an intelligent interpretation of the cardiogram may afford diagnostic evidence of some value. For instance, the relative lengths of the diastolic and systolic periods of the ventricular cycle may be gauged by this means with some accuracy. In mitral stenosis and in dilatation of the left ventricle the diastole is relatively prolonged, whereas in aortic regurgitation and in hypertrophy of the ventricle the same period is relatively shortened, and hence the cardiographic evidence in the differential diagnosis of these affections may be requisitioned with advantage.

In describing a cardiogram it is perhaps simplest to make use of the same nomenclature as was employed in the account of the sphygmogram.

Thus a cardiogram may be said to consist of an ascending limb or upstroke ($k-d$), an apex (ade), and a descending limb or downstroke ($d-g$).

kla = diastolic rise ; $a-d$ = systolic rise

$d-f$ = systolic fall ; $f-k$ = diastolic fall

The Ascending Limb

It will be noticed that this portion of the curve comprises both diastolic and systolic rises. The first part of the diastolic rise generally shows an elevation (k) which marks the filling of the ventricle by its active dilatation. This elevation is particularly well marked when the dilatation of the chamber is energetic as in (1) hypertrophy of the heart with powerful suction action on dilatation ; (2) low degrees of blood tension with rapid systole and sudden relaxation of the ventricle.

Immediately following the elevation k is a gradual ascent of the upstroke l . It represents the passive filling of the ventricle between the first inrush of blood, due to its active dilatation, and the final act of filling by the auricular systole a . The steepness and length of this part of the curve depend largely on the total length of diastole ; when diastole is prolonged this section of the curve is extensive and the rise gradual. When diastole is short the opposite conditions obtain. The diastolic rise sometimes shows elevations in addition to k and a . These represent irregular contractions of the auricle, or are due to vibrations of the auriculo-ventricular valves.

The elevation a at the end of the diastolic rise is due to the auricular systole, and is specially well marked when the left auricle is hypertrophied as in mitral stenosis.

As a rule the elevation a is followed by a notch, which separates the diastolic from the systolic rise. Sometimes, however, this notch is absent, in which case the diastolic and systolic rises are continuous. The systolic rise varies in amplitude in different cases. Its height, however, depends more on the suddenness of the ventricular systole than on its force. Thus in palpitation and cases of low-tension

pulse the amplitude of this portion of the tracing may be very great, whereas in hypertrophy the rise may be comparatively small.

The Apex

The degree of acuteness of this angle depends chiefly on the suddenness with which the ventricle empties itself, and is due to the falling away of the heart's apex from the chest wall. It is especially acute in mitral regurgitation, in which condition the ventricle has two outlets by which it can empty itself, viz. through the incompetent mitral valve and through the aortic orifice.

The Descending Limb

f marks the completion of the systole, and the distance ($d-f$), i.e. the systolic fall, is a rough measure of the length of the systole. In hypertrophy of the heart, and in aortic stenosis, this section of the curve is of considerable extent, and the apex is usually more or less rounded.

$f-k$, the diastolic fall, marks the beginning of diastole immediately after the closure of the semilunar valves. It is usually nearly perpendicular, and occupies, consequently, a very short period before the filling of the ventricle causes the diastolic rise.

The characteristic features of the cardiograms taken from cases representing the chief forms of valvular disease will now be briefly considered.

MITRAL STENOSIS

The diastolic rise is usually prolonged, and the elevation in it due to the auricular systole is well marked. There may be secondary elevation on this limb of the cardiogram, due to the causes enumerated above.

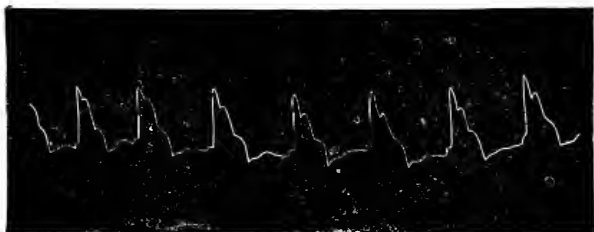


FIG. 30. CARDIOGRAM FROM A CASE OF MITRAL STENOSIS
(Sansom)

In this tracing the diastolic rise is of considerable length and inclined obliquely upwards, showing the long period occupied in

the filling of the ventricle through the narrowed mitral orifice. Secondary undulations are present on this section of the tracing, due to irregular contractions of the auricle, or to vibrations set up at the mitral valve. The elevation due to the auricular systole is not sufficiently well marked to suggest great hypertrophy of the left auricle.

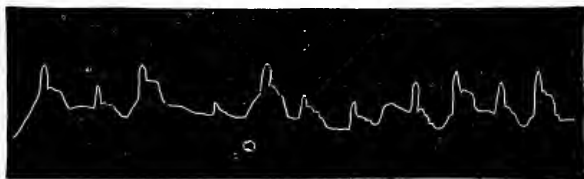


FIG. 31. CARDIOGRAM FROM A CASE OF MITRAL STENOSIS
(Sansom)

This tracing shows great irregularity of the heart's action, which is due to the supervention of heart failure with tricuspid regurgitation.



FIG. 32. CARDIOGRAM FROM A CASE OF MITRAL STENOSIS
(Sansom after Galabin)

This tracing shows a great elongation of the "diastolic rise" section of the ascending limb.

MITRAL REGURGITATION



FIG. 33. CARDIOGRAM FROM A CASE OF MITRAL REGURGITATION
(Sansom)

This cardiogram shows great irregularity of the heart's action. The length of the diastolic rise is very variable, and the rise due to the auricular systole is badly marked. The apex is bifid, the

second elevation being due to the contraction of the musculi papillares (Roy and Adami).

AORTIC STENOSIS



FIG. 34. CARDIOGRAM FROM A CASE OF AORTIC OBSTRUCTION
(Sansom after Galabin)

Owing to the prolonged ventricular contraction the systolic rise is gradual and the apex rounded. The total systolic period is lengthened, and the diastolic rise short.

AORTIC REGURGITATION

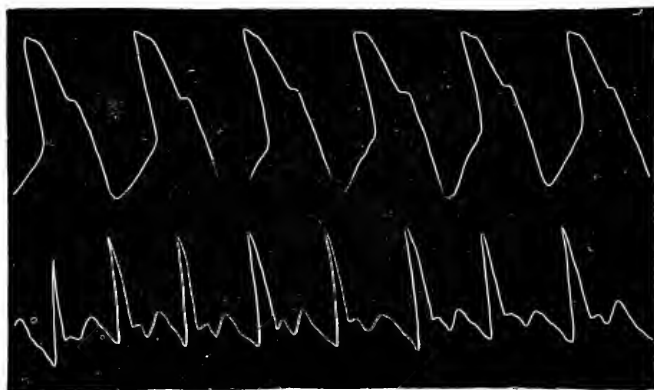


FIG. 35. CARDIOGRAM AND SPHYGMOGRAM FROM A CASE OF
AORTIC REGURGITATION
(Sansom)

The diastolic rise is steep, owing to the rapid filling of the ventricles due to the regurgitation. The systolic rise is steep, owing to the sudden contraction of the ventricle. The apex is acute, and the systolic fall steep.

CHAPTER VI

CONGENITAL AFFECTIONS OF THE HEART AND GREAT VESSELS

Ætiology and Pathology—Classification—Symptoms—Physical Signs—Diagnosis
—Prognosis—Treatment.

ÆTIOLOGY AND PATHOLOGY

MALFORMATIONS and misplacements of the heart represent, for the most part, permanent survivals of foetal conditions. A short account, therefore, of the development of the cardio-vascular system, in so far as it bears on this subject, will render the mode of origin of the congenital affections of the heart and great vessels more intelligible to the reader.

At an early period of development the heart consists of two separate tubes of mesoblast, which coalesce in the median line soon after the thirteenth day, to form in the upper part of the neck a tube of single bore, which is slightly twisted on itself. As development proceeds this tube loses all traces of its original double bore, and during the third week a constriction appears which marks the primitive division into auricle and ventricle, the former lying somewhat behind (*i.e.* posterior) to the latter. At the same time the tube becomes further removed from the cephalic end of the foetus, and gravitates as a whole towards the position it finally occupies in the thorax.

The tube then becomes twisted upon itself in such a way that the auricular portion comes to lie behind and below the ventricular portion. The single auricle receives the two venæ cavæ, and the single ventricle gives rise to the common arterial trunk (*truncus arteriosus*).

The constriction between the primarily undivided auricle and ventricle appears internally as a ridge of tissue (the primitive auriculo-ventricular septum), which, by means of two localized outgrowths (the endocardial cushions) from points on opposite sides of the tube, effects a median junction, with the result that two passages are formed—the right and left auriculo-ventricular openings.

The auricle undergoes division before the ventricle.

The septum first appears at the upper and posterior part of the auricular cavity, and the free edge grows forwards and downwards to join with the fused cushion-like thickenings which divide the auriculo-ventricular orifices, and also form part of the interventricular septum.

Before the completion of the auricular septum a new orifice, the foramen ovale, makes its appearance at the upper and posterior part of this structure. The closure of the foramen ovale is effected at a later period by the development of a second septum, which also grows from the superior auricular wall, a little to the right of the original partition. The new septum grows forward, and acts as a valve or curtain, which is gradually drawn over the foramen ovale, and at birth completely closes the opening.

The septum atriorum is situated to the left of the orifices of the superior and inferior venæ cavæ, and hence these vessels discharge their contents into the right auricle. The pulmonary veins are, at an early date, found in connection with the left auricle.

The ventricle subsequently undergoes division into two cavities by the evolution of the interventricular septum, which has a triple origin, namely, from (1) a muscular upgrowth from the antero-inferior part of the primitive ventricle; (2) the endocardial cushions, which divide the common auriculo-ventricular orifice into a right and left half; and (3) the septum, which divides the common arterial trunk into an aortic and pulmonic moiety. The interventricular septum is completed by the fusion of these three segments. It is the anterior part of the septum, viz. the portion which lies between the two arterial orifices, which is most commonly the seat of incomplete development. Less frequently the posterior segment, which is situated between the two auriculo-ventricular openings, is partially or entirely absent.

The membranous portion of the septum is the part which lies between the anterior and posterior sections. It has been called the undefended space by English authors.

This membranous portion has been regarded as the part of the septum which is most liable to defects of development. Rokitsansky has, however, conclusively shown that of all sections of this structure it is the least liable to malformation.

DEVELOPMENT OF THE GREAT VESSELS IN RELATION WITH THE HEART

The two primitive tubes which coalesce and constitute the heart continue fused together for a short distance beyond this viscus, and form the common arterial trunk or conus arteriosus.

At a later period this vessel becomes differentiated into the aorta and pulmonary artery by the development of a longitudinal septum, which divides the originally single tube into two portions. This septum ultimately effects a junction below with the interventricular

and interauricular septa in such a way that it not only completes the separation of the ventricles, but also cuts the connection between the aorta and right ventricle, and places this vessel exclusively in

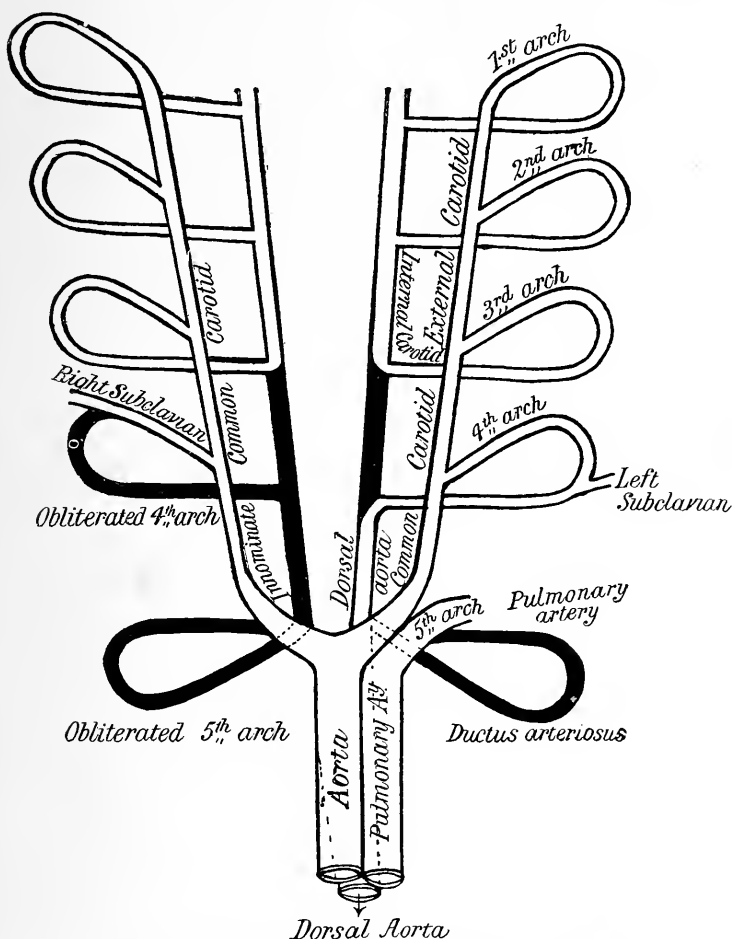


FIG. 36. THE AORTIC ARCHES, SHOWING MODE OF TRANSFORMATION INTO PERMANENT ARTERIAL TRUNKS. THE PARTS IN BLACK BECOME OBLITERATED (After MacAlister, modified)

communication with the left ventricle. Above, the disposition of the septum is such that it directs blood coming from the right ventricle into the ductus arteriosus, or fifth arch, leaving a small communication only between the aorta and pulmonary artery.

Finally this opening is also closed, so that at birth the right ventricle pours the whole of its contents into the pulmonary artery, whereby its area of distribution is confined to the lungs.

The two primitive tubes beyond the conus arteriosus continue as separate structures towards the cephalic end of the foetus, and on each side supply in succession branches which ascend between the visceral clefts, and unite again at the posterior extremity of these clefts to form a single vessel on each side. These two vessels, the primitive descending aortæ, run down the trunk on either side of the notochord, giving off as they descend lateral branches to the body wall and yolk sac, and terminate in two large vessels which accompany the allantois, and furnish blood to the foetal portions of the placenta.

The two primitive descending aortæ at an early period of development coalesce immediately behind (*i.e.* posterior to) the heart to form a single tube, which runs in the median line up to its bifurcation into the two common iliac arteries. Above their point of junction the two primitive vessels remain as single tubes on either side of the notochord. The tube on the right side ultimately disappears, while the one on the left persists as the first part of the descending aorta, and becomes directly continuous below with the single fused vessel (Fig. 36).

The branches or arches, as they are called, which arise on either side from the two primitive vessels forming the continuation of the truncus arteriosus and pass between the visceral clefts, are five in number. The first three are not of any interest, so far as the subject under discussion is concerned. The destinations of the fourth and fifth arches are, however, of great importance, as they bear directly on the question of cardiac malformations.

The fourth arch on the right side forms the arteria innominata and root of the right subclavian artery. On the left side it persists as the aortic arch and first part of the descending aorta. The fifth arch on the right side becomes obliterated. The fifth left arch, or ductus arteriosus, persists throughout its entire length during intra-uterine life. It joins the continuation of the fourth arch on the left side, and is therefore continuous with the descending aorta and umbilical arteries. Moreover, since the ductus arteriosus, through the pulmonary artery, is in direct continuity with the right ventricle, the foetal portions of the placenta are chiefly supplied with blood from the right heart. At birth, with the expansion of the lungs, the distal connection of the ductus arteriosus with the aorta becomes obliterated, and forms a fibrous cord, the ligamentum arteriosum.

The proximal end of the ductus arteriosus, however, persists as the pulmonary artery.

In the light of the developmental connections of the great vessels, it is obvious that in cases of pulmonic stenosis, or in conditions affecting the patency of the pulmonary artery during foetal life, the blood supply to the lungs after birth can be supplemented

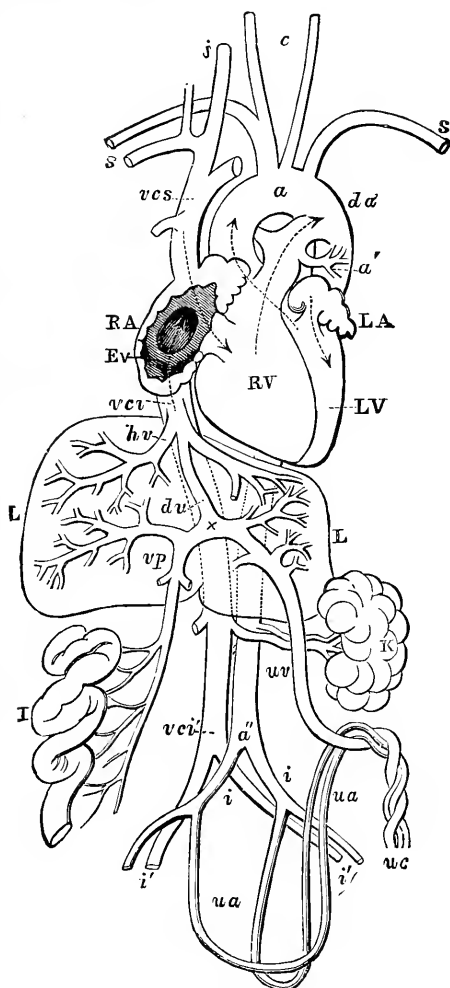


FIG. 37. DIAGRAMMATIC OUTLINE OF THE ORGANS OF CIRCULATION
IN THE FŒTUS OF SIX MONTHS

(Thompson, *Quain's Anatomy*)

RA=right auricle; RV=right ventricle; LA=left auricle; Ev=eustachian valve; LV=left ventricle; L=liver; K=left kidney; I=portion of small intestine; *a*=arch of aorta; *a'*=its dorsal part; *a''*=its lower end; *vcs*=superior vena cava; *vci*=inferior vena cava where it joins the right auricle; *vci'*=its lower end; *s*=subclavian vessels; *j*=right jugular vein; *c*=common carotid arteries; four curved dotted arrow lines are carried through the aortic and pulmonary opening and the auriculo-ventricular orifices; *da*, opposite to the one passing through the pulmonary artery, marks the place of the ductus arteriosus; a similar arrow line is shown from the vena cava inferior through the fossa ovalis of the right auricle and the foramen ovale into the left auricle.

by way of the aorta through either extremity of the ductus arteriosus.

Conversely, the blood current through the aorta, in case of obstruction at the aortic orifice or in the course of the vessel, can be reinforced by way of the pulmonary artery or its continuation, the ductus arteriosus (Fig. 37).

In want of a more scientific explanation, the malformations of the heart and great vessels are commonly ascribed to congenital errors of development, or to hereditary deficiency of developmental power. Expressions of this kind avoid physical explanations, and are little more than admissions of our ignorance of the true causes of congenital cardiac anomalies. It is probable, however, that when more than one member of the same family has been the subject of a similar cardiac malformation there has been the same mechanical cause at work, although it is not possible, in the present state of our knowledge, to demonstrate its nature.

Maternal impressions have also been held responsible for a considerable number of cardiac malformations, a belief which is founded more on popular superstition than on scientific reasoning.

On the other hand, there are a considerable number of cases of cardiac mal-development for which we can assign approximate physical causes, although it may be that in some of these instances a link here and there in the chain of argument is forged on a theoretical hypothesis.

After birth, the strain which may be imposed on the aortic and mitral valves by increased arterio-capillary resistance, and the variable demands made upon the left side of the heart by alterations in blood pressure, not only predispose to valvular inflammation, but also frequently induce structural changes in the cavity and wall of the left ventricle. If organic changes of this character can be wrought in the completely developed heart, it is hardly surprising that similar conditions operating, in utero, on a heart which is in process of evolution should evoke alterations as profound and extensive as those in clinical records.

The foetus, as it lies in utero, is to a large extent protected from disturbances of blood pressure, so far as its intrinsic circulation is concerned. Nevertheless, the circulation through the umbilical cord, as well as through the foetal portion of the placenta, must, in the ordinary course of events, be exposed to considerable variations in blood pressure. It has already been shown that the umbilical arteries are branches of the descending aorta, which for the most part receives its blood supply, through the ductus arteriosus, from the right ventricle. Hence, in the foetus it is the right side of the heart, with its included valvular mechanisms, which bears the brunt of the circulation. Consequently we should expect that a departure from the normal course of development would be more liable to occur on the right than on the left side of the heart. Statistics bear out this reasoning, for out of 181 cases of cardiac

malformation collected by Peacock 119 were associated with lesions at the pulmonic orifice.

Furthermore, if it be admitted that defects of development may be produced at the pulmonic orifice by mechanical causes in the manner that has been suggested, it is comparatively easy to assign a physical reason for other and secondary structural imperfections which may be found in association with these lesions.

The abnormalities of development to which attention will be especially directed are those affecting (1) the interventricular septum, (2) the interauricular septum, (3) the junction of the aorta with the right or left ventricle, (4) the patency of the ductus arteriosus.

If the pulmonary artery or its orifice become the seat of obstruction, so that the passage of blood through the vessel is interfered with, before the division of the ventricles is completed by the junction of the septum of the conus arteriosus with that of the ventricles, the increase of pressure in the right ventricle, by causing a flow of blood from one side of the heart to the other during systole, may delay or altogether prevent the union of these two segments of the interventricular partition. Under these circumstances the aorta may not only communicate with the left, but also with the right ventricle through the deficiency in the septum. Moreover, this condition may become a permanent one, so that as long as life is maintained both the aorta and pulmonary artery are conjointly fed by the right and left ventricles.

The septum of the ventricles may, however, be partially incomplete, although the septum of the conus arteriosus has fused with that of the ventricles, and thus established direct communication between the aorta and left ventricle and between the pulmonary artery and right ventricle. In such an event, if stenosis of the pulmonic orifice occur, the blood (owing to the rise of pressure in the right ventricle consequent on the difficulty in the discharge of its contents) takes the path of least resistance through the opening in the septum into the left ventricle. The constant irritation and disturbance thus produced will delay or prevent the completion of the interventricular septum.

Again, the obstruction to the passage of blood through the pulmonic orifice may arise after the completion of the interventricular septum, but while there is still a communication between the two auricles through the foramen ovale. In this event the increase of pressure in the right ventricle gives rise to more or less regurgitation into the right auricle, so that part, at least, of the blood current is diverted into the left auricle through the patent foramen ovale.

If the degree of pulmonic stenosis is moderate, the right ventricle hypertrophies, owing to the difficulty experienced in the discharge of its contents; the left ventricle hypertrophies from overwork, and the ductus arteriosus may or may not remain patent after birth.

If the degree of pulmonic stenosis is very great, the right ventricle

ultimately atrophies, and is practically thrown out of the circulation; the left ventricle hypertrophies from overwork, and after birth supplies the lungs with blood through the aorta and patent ductus arteriosus.

In like manner, obstruction on the left side of the heart determines corresponding anomalies of development.

Stenosis of the aortic opening may divert the blood flow through the imperfect septum of the auricles or ventricles into the right side of the heart, and thus, by constant irritation, prevent the perfect evolution of these structures. The right side of the heart then carries on both the systemic and pulmonic circulations, the pulmonary artery dividing its contents between the ductus arteriosus and aorta above the seat of obstruction.

Deficiency of the interauricular or interventricular septum is not only compatible with life, but also with a healthy, well-organized life, so soon as the heart becomes balanced to the altered conditions of the circulation.

It has been shown, therefore, how obstruction at one or other of the orifices of the heart may determine the further course of development. In the instances which have been given, the obstruction has occurred at a somewhat late period in the evolution of the organ. There is no reason why interference with the circulation through the heart and great vessels should not occur at earlier periods, and exercise an influence similar in kind but more far-reaching in its effects, and thus determine errors in development for which we can otherwise find no satisfactory explanation.

In the attempt which has been made to show that abnormalities in the development of the heart depend in a large measure on mechanical causes, no mention has been made of demonstrable inflammatory conditions of the valves and endocardium. Endocarditis does, however, occasionally occur in intra-uterine life, and gives rise, for the most part, to stenosis of the orifice, which is the seat of the inflammatory process. The effects of the lesion on the further course of the development of the heart are similar in kind to those produced by stenosis of the orifices from other causes.

The form of endocarditis most commonly observed is the chronic or sclerotic variety. It usually occurs on the right side of the heart, for two reasons: firstly because the right side of the heart is exposed to greater strain than the left, and secondly because malformations occur more commonly on the right side of the organ, and inflammatory conditions appear to become more readily superimposed on imperfectly, than on perfectly developed structures.

The exciting causes of foetal endocarditis are probably blood conditions existing in the mother, which are transmitted to the embryo through the foetal circulation.

CLASSIFICATION

Congenital affections of the heart occur either as misplacements or malformations of the organ.

MISPLACEMENTS OF THE HEART

1. **Dextro-cardia** is the condition in which the heart occupies a position in the chest which may be described as the "negative" of its ordinary position.

In its relation to the middle line the heart normally lies obliquely with its apex pointed to the left. In the condition under discussion the organ points to the right. The heart should lie not presenting its two sides in equal degree to the front, but turned on its axis, so that the right ventricle looks forwards, and the left backwards. In the condition of dextro-cardia, the left ventricle looks forwards and the right ventricle backwards. The heart should not lie in the middle vertical line of the body, but should be placed somewhat tilted forwards, so that its apex lies in advance of its base. In the abnormal condition this relation is still maintained.

The original position of the heart in the embryo is in the course of development normally altered by twists in three directions.

From the vertical middle line it is twisted to the left; on its vertical axis it is twisted to the left; on the horizontal line it is twisted to the front. In the abnormal condition under discussion the two first-mentioned twists are reversed, while the third remains as in the normal state.

Clinically, the result of this abnormality is that the apex of the heart is found on the right instead of the left side of the chest, and the bulk of the organ is situated to the right instead of to the left of the median line. Nothing more can be discovered at the bedside, but, post-mortem the aorta and pulmonary artery are also reversed in their directions.

Dextro-cardia may be found as an independent condition, but it is usually associated with transposition of the other viscera. Thus the liver is on the left side, the stomach and spleen on the right. The cæcum is on the left, the sigmoid flexure on the right, while the left lung has three lobes, and the right two only. No symptoms whatever result from this condition, but its clinical importance arises from the liability to mistake the misplacement of the heart and viscera for other conditions, the outcome of disease.

2. **Mesocardia**.—In this condition the heart occupies a central position in the thorax, a situation normal to it in the early stages of development.

3. **Ectopia cordis**.—This abnormality occurs in three forms—

1. Ectopia cervicalis
2. Ectopia pectoralis
3. Ectopia abdominalis

1. *Ectopia cervicalis* is an extreme case of misplacement, in which the heart occupies a position in close proximity to the lower jaw. The organ may be exposed or covered with a layer of skin.

2. *Ectopia pectoralis* is usually associated with more or less deficiency of the sternum and thoracic wall. The heart may be freely exposed, or covered by its pericardium only, or by skin and pericardium.

3. *Ectopia abdominalis* is the form in which the heart lies, below the diaphragm, in the abdominal cavity.

The two first-mentioned varieties of ectopia cordis are hardly compatible with extra-uterine life. In the third variety life has been maintained for some years.

MALFORMATIONS OF THE HEART

Deficiency of the pericardium.—Complete absence of the pericardium is exceedingly rare as an independent condition.

In most instances complete absence of the pericardium is found in association with ectopia cordis. Partial defect is sometimes observed.

Abnormalities of the cardiac wall.—Hypertrophy or atrophy of one or other of the chambers of the heart is not uncommonly found. The only other abnormality in this class that requires notice is the condition known as divided or bifid apex, which has no clinical interest.

Anomalies of the cardiac septa.—In very rare instances both the interauricular and interventricular septa are entirely absent. The heart then consists of two cavities, and the condition is termed “cor biloculare.”

In still rarer cases the interauricular septum persists while the interventricular septum is completely absent, so that the heart consists of three cavities, the so-called “cor triloculare.”

Complete absence of the interauricular septum with persistence of the interventricular septum is extremely rare.

Interventricular septum.—Partial deficiency of this septum is not uncommon. The deficiency usually affects the hinder part of the anterior section just in front of the membranous portion of the partition. Less commonly the posterior part of the septum is deficient. In rare instances the membranous portion is found wanting.

Interauricular septum.—Partial defects of this septum are not infrequently observed. The abnormalities which may be found are—

1. Absence of the anterior muscular portion of the septum.
2. Complete patency of the foramen ovale.
3. Incomplete closure of the foramen by the valve.
4. Perforations round the edge of the membrane which closes the foramen.
5. An oblique opening beneath the edge of the valve when this membrane is not united to the anulus.
6. The membrane which should close the foramen may be more or less cribriform in structure.

ANOMALIES OF THE VALVES OF THE HEART

The Semilunar Valves

These valves may consist of two cusps only, or by the development of supernumerary segments their number may be increased to four or five.

The pulmonic orifice and artery.—The following forms of obstruction at this orifice, and in the course of the vessel, are found :—

1. Stenosis of the pulmonic orifice frequently found in association with defects of the cardiac septa.
2. Atresia of the orifice and first part of the pulmonary artery.
3. Stenosis of the trunk and canal of the pulmonary artery.
4. Stenosis of the conus arteriosus. In this event the infundibular portion of the right ventricle is the part chiefly affected.

The aortic orifice and aorta.—The following abnormalities are observed :—

1. Stenosis of the aortic orifice.
2. Atresia of the orifice and first portion of the aorta.
3. Stenosis of the arch of the aorta at the ductus arteriosus. This is a very rare condition, and is due to a too great extension of the cicatricial process involved in the closure of the ductus arteriosus.

The Auriculo-Ventricular Valves

The mitral valve may consist of three flaps. The edges of the curtains of the valve may be united to form a continuous membrane with a central aperture.

The tricuspid valve may consist of four or more flaps. The edges of the curtains may be adherent with the production of stenosis of the orifice.

Anomalies of the tricuspid valve frequently coexist with defects of development at the pulmonic orifice.

ANOMALIES OF THE LARGE VESSELS

1. Transposition of the aorta and pulmonary artery, either with or without transposition of the mitral and tricuspid valves, is sometimes observed.
2. The septum between the aorta and pulmonary artery may be wholly or partially deficient, so that the two vessels communicate with each other.

3. Persistence of the ductus arteriosus.

The closure of this vessel usually takes place within a few days after birth. If the normal process of involution fails, a communication is left between the aorta and the left branch of the pulmonary artery. The result is more or less regurgitation from the aorta into the lungs. Patency of the ductus arteriosus is usually associated with other developmental anomalies of the heart.

4. Premature closure of the ductus arteriosus.

This condition is usually associated with imperfect development of the pulmonary artery.

Pathologically the most common congenital anomalies of the heart are stenosis of the pulmonary artery, defects of the cardiac septa, and abnormalities of the sigmoid valves.

SYMPTOMS

The symptoms which arise in connection with congenital affections of the heart are referable, for the most part, to derangements of the circulation.

The earliest and most characteristic feature of congenital disease of the heart is cyanosis. It occurs in about 90 per cent. of the cases, and the degree of its manifestation varies between a slight lividity and a dark purple discoloration (see p. 23). The extremities, the ears, nose, and face are the parts through which the circulation is maintained with the least facility, and consequently it is in these situations that the cyanosis is most marked.

If the cardiac lesion is compatible with life, the further development and nutrition of the tissues in general, and of the above-mentioned parts in particular, are seriously interfered with. Growth is stunted, the fingers and toes become club-shaped, often in association with a claw-like appearance of the nails, and unhealthy sores may arise in various parts of the body.

It is somewhat anomalous that dropsy is seldom observed with congenital affections of the heart, although the conditions which are apparently essential for its production are present in many instances.

Attacks of palpitation and cardiac distress are of frequent occurrence, and are apt to be excited and exaggerated by emotion, exposure to cold, etc. The inability of the heart to cope with the circulatory difficulties is shown also by rapid and laboured breathing, cough, etc., and sometimes by attacks of dyspnoea, which may culminate in convulsions.

Inflammatory affections of the lungs, hæmoptysis, and tubercular disease are likewise predisposed to by the continued derangement of the pulmonic circulation.

The nervous and digestive systems are not exempt from the effects of the general circulatory incompetence.

Cerebral hæmorrhage, attacks of convulsions or unconsciousness, lethargy of mind, gastric and intestinal disturbances, are often referable to interference with the blood supply of these systems.

The temperature of the internal parts is usually normal, while that of the surface of the body is, as a rule, considerably lowered, in consequence, no doubt, of the loss of heat by the dilated capillaries and the sluggish circulation through them.

PHYSICAL SIGNS

Physiognomy and general appearance.—The aspect and general appearance of the patient, which in the large majority of cases is highly suggestive of insufficient aëration of the blood, has already been described.

Gibson has recently called attention to the remarkable concentration of the blood which obtains in cases of congenital disease of the heart. Examination shows an increase in the specific gravity, as well as in the hæmoglobin and corpuscular ingredients of the blood. Gibson attributes the condition to the lessened wear and tear of the red blood cells, consequent on the diminished metabolism that takes place with venous stasis.

Pulse.—The pulse does not present, as a rule, any features of special interest. It is frequently irregular both in force and frequency, and these characters are especially noticeable during the attacks of palpitation to which the subjects of congenital disease of the heart are liable. In cases of pulmonic stenosis the size and strength of the pulse are said to be of service in the determination of the condition of the interventricular septum. Thus if the pulse be small in the radial artery, the inference is that the supply of blood to the left ventricle is diminished, and hence the septum is perfect.

On the other hand if the pulse in the radial artery be of normal volume and strength, the inference is that the supply of blood to the left ventricle is supplemented from some other source, and hence the probability of the existence of an imperfect septum ventriculorum.

The tone of the arterial system is, generally speaking, below par.

Heart.—Inspection commonly reveals prominence or bulging of the chest wall in the præcordial area, involving the lower part of the sternum and third, fourth, and fifth left costal cartilages and intercostal spaces.

Pulsation is often visible in these situations, but, as a rule, is most marked in the epigastrium.

The cardiac impulse is forcible and displaced outwards.

A thrill systolic in time is sometimes palpable over the base of the heart at or about the level of the second left intercostal space.

Percussion may demonstrate an increase in the area of cardiac dulness, either to the right or left of its normal limits.

The auscultatory phenomena depend on the site and nature of the lesion.

Pulmonary stenosis gives rise to a loud blowing systolic murmur, which is most distinctly heard over the second left costal cartilage, though in many instances it is audible over the greater portion of the præcordial area. The second sound may be weak or accentuated, and is sometimes accompanied or followed by a diastolic bruit.

Patency of the ductus arteriosus is, in all probability, indicated by a loud rumbling systolic murmur, most clearly audible at or slightly to the left of the pulmonic cartilage, in conjunction with accentuation of the pulmonary second sound.

Deficiency of the interventricular septum occasions a very loud and more or less musical systolic, or sometimes nearly continuous bruit, which is very commonly audible over the greater portion of the chest. The characteristic feature of the murmur is that it is heard most distinctly along the course of the interventricular septum and rapidly diminishes in intensity as the stethoscope leaves this line (Maguire).

The direction of the interventricular septum is approximately shown by a line drawn from the second left costal cartilage at its junction with the sternum to a point about half an inch inside the apex beat. So far as is known, deficiency of the interauricular septum in the form of patent foramen ovale gives rise *per se* neither to symptoms nor physical signs of any importance.

The physical signs produced by congenital lesions at the aortic orifice do not differ from those found in association with the similar acquired forms of disease at this orifice.

DIAGNOSIS

The differential diagnosis of congenital and acquired disease of the heart seldom presents much difficulty, provided a careful consideration be given to the history and physical signs of the case, including an examination of the blood.

The diagnosis of the precise nature of the congenital defect, or defects, is a much more complicated problem, and one that in many instances it is impossible to elucidate.

Very extensive cardiac malformations are usually incompatible with the duration of life for more than a few hours or days, hence, except during the earliest periods of extra-uterine life, the differential diagnosis of congenital lesions of the heart is restricted within comparatively narrow limits.

From a clinical point of view, chief interest centres in the diagnosis of pulmonary stenosis, since in the immense majority of cases this lesion is found in patients, presenting a history of cyanosis from the time of birth in association with other indications of a malformed heart, who have survived the twelfth year (Peacock). If, therefore, in a patient over twelve years of age, with a history of cyanosis since birth, there is evidence of enlargement of the right ventricle in

addition to the presence of a systolic bruit in the pulmonic area, which is not conducted up into the neck, a diagnosis of pulmonary stenosis is certainly justifiable.

The presence or absence of associated deficiency of the interauricular or interventricular septum, or of an open state of the ductus arteriosus, or of some combination of these conditions, cannot, as a rule, be determined with any degree of certainty. (See p. 111.)

Since patency of the foramen ovale is unaccompanied by any known symptoms or physical signs, the question of the presence or absence of this anomaly does not require any further consideration.

Deficiency of the interventricular septum will be indicated by enlargement of both ventricles and by the presence of a systolic, or of a more or less continuous murmur, which can be heard most distinctly in the mid-sternal region and along the course of the interventricular septum.

A murmur having these characters should suffice to distinguish deficiency of the interventricular septum occurring as an independent lesion from other forms of congenital disease of the heart.

If, in association with the symptoms and signs of pulmonic stenosis, the amount of hypertrophy of the right ventricle were much greater than the degree of pulmonary narrowing, estimated on other grounds, would account for, the existence of a patent condition of the ductus arteriosus may be suspected. This suspicion would be strengthened by the presence of accentuation of the pulmonic second sound.

The symptoms and signs of congenital affections of the other cardiac orifices and valves do not call for special comment, inasmuch as they resemble for the most part those found in connection with similar lesions of acquired origin. Nevertheless, it must be borne in mind that the frequent concurrence of two or more congenital anomalies of the heart must of necessity lead to more or less overlapping and modification of the symptoms and signs due to the respective lesions.

Transposition of the great vessels is sometimes found in association with transposition of the heart and other viscera, but *per se* this abnormality does not give rise to any diagnostic symptoms or signs.

PROGNOSIS

Dextro-cardia, meso-cardia, small openings in the septa of the heart, and malformations of the valves exercise but little influence on the ordinary expectancy of life, except in so far as the last-mentioned condition may predispose to the incidence of inflammatory affections of these structures.

Moderate stenosis of the trunk or orifice of the pulmonary artery, without other malformation, but with adequate compensatory hypertrophy of the right ventricle, is compatible with the enjoyment of

many years of healthy life, and old age has more than once been attained under these conditions.

In point of fact, if the unimportant lesions mentioned above be excluded, the prognosis in uncomplicated pulmonary stenosis is more favourable than in any other form of congenital cardiac affection.

If patency of the foramen ovale is combined with narrowing of the pulmonic orifice, apart from the fact that this association usually implies a greater degree of stenosis of the arterial opening, the expectancy of life is materially curtailed. Nevertheless, in exceptional cases of this kind the patient has reached middle life, but, as a rule, the age at death seldom exceeds fifteen or twenty years.

The duration of life is still further reduced if the pulmonic narrowing is associated with deficiency of the interventricular septum, for under these conditions the patient seldom reaches the age of puberty.

If the pulmonic lesion is combined with an open state of the ductus arteriosus, life is rarely prolonged beyond the thirteenth year.

When the pulmonary orifice or artery is completely impervious, the patient does not, as a rule, survive the first two or three years.

With regard to stenosis of the aortic orifice, the prospect of life depends largely on the degree of narrowing, and on the concomitant lesions. If the stenosis has been sufficient to establish patency of the interventricular septum, life is seldom prolonged beyond the tenth year.

When the heart consists of one ventricle, with a double or single auricle, the expectation of life can usually be expressed in days rather than years, although a few cases are recorded of survival beyond infancy.

Transposition of the aorta and pulmonary artery is incompatible with any lengthened period of existence. Patency of the ductus arteriosus is usually combined with stenosis of the pulmonic or aortic orifice. The prognosis in these cases depends largely on the degree of narrowing which obtains at the arterial openings. When patency of the ductus arteriosus occurs as an independent lesion, it does not appear to have a very unfavourable influence on the duration of life, since the subjects of this malformation have in several instances reached middle age.

Patent foramen ovale does not *per se* seriously interfere with the duration of life.

During infancy the cause of death is frequently traceable to mechanical interference with the circulation. A large number of the subjects of congenital cardiac disease die from cerebral or pulmonary complications.

If the patient reaches adult life, the most common cause of death is tubercular disease of the lungs. In some instances the fatal termination is brought about by cardiac failure.

TREATMENT

The treatment of congenital affections of the heart should be conducted on the general principles, which will be considered in some detail under the section dealing with the acquired varieties of morbus cordis. It will therefore be unnecessary here to give more than a brief résumé of the treatment that is especially applicable to congenital heart disease.

It has already been pointed out that the symptoms are referable, for the most part, to disturbance of the circulation. All measures, therefore, which increase the effectiveness of the heart's action, or relieve obstruction to the onward flow of blood, have their therapeutic value in the treatment of congenital disease of the heart.

Hygienic treatment, which is of the first importance, comprises rest, the avoidance of excitement or exertion, warmth, fresh air, and a carefully regulated diet. All conditions liable to induce bronchial irritation should be sedulously avoided. Careful protection against cold, and the adoption of an open-air life, would go far to counteract the liability to pulmonary tuberculosis that is observed after adult age is reached.

Recourse may be had to massage, with the object not only of relieving superficial congestion and of assisting the onward flow of blood, but also of restoring tone to the tissues and bloodvessels without the intervention of active exercise.

Medicinal treatment, except in so far as depletion of the vascular system can be accomplished by saline purgatives and diuretics, consists in the careful exhibition of cardiac tonics and in the relief of complications.

Digitalis must be used with caution, but under suitable conditions it is often of great service, more especially in combination with strychnine.

Hypodermic injections of ether are of value when the use of digitalis is contra-indicated.

Attacks of dyspnoea and palpitation, which are often very troublesome at night, may be treated by the compound spirits of ether, by camphor and spirits of chloroform, or by alcohol, in the form of whisky or brandy. In urgent cases of this kind, and in convulsive seizures, relief may be obtained by the direct abstraction of blood, which can be effected by means of leeches, or by venesection. It must not, however, be forgotten that removal of blood, except in very small quantity, is not well borne by the subjects of congenital disease of the heart.

CHAPTER VII

DISEASES OF THE PERICARDIUM

Classification—Section I. Acute Pericarditis—Section II. Pericardial Adhesion—Section III. Hydropericardium—Section IV. Hæmopericardium—Section V. Pneumopericardium—Section VI. New Growths.

THE morbid conditions which may effect the pericardium will be considered under the following arrangement :—

1. Acute pericarditis
2. Pericardial adhesion (including chronic pericarditis, external pericarditis, etc.)
3. Hydropericardium
4. Hæmopericardium
5. Pneumopericardium
6. New growths

SECTION I

ACUTE PERICARDITIS

ÆTIOLOGY

Acute inflammation of the pericardium occurs most commonly in association with rheumatism and Bright's disease, and is found about twice as frequently in connection with the former as with the latter disorder.

As a rule it appears early in rheumatism and late in Bright's disease, and is a much less fatal complication of the one than of the other.

Pericarditis sometimes arises in the course of enteric fever, scarlatina, measles, small-pox, septicæmia, and pyæmia. It may occur, too, as the result of the extension of an inflammatory or other morbid process from neighbouring structures, such as the heart; pleuræ, lungs, mediastina, and their glands; liver; stomach; peritoneum, etc. It is occasionally due to direct mechanical injury, as, for instance, may be caused by a blow in the præcordial area. Inflammation of the pericardium is not infrequently associated with pneumonia, especially in children. In this connection the pericardial affection may be due to the extension of the inflammatory process from the lungs, or to toxæmic causes.

Among the many micro-organisms which are found in connection with pericarditis may be mentioned streptococci, staphylococci, micrococci, pneumococci, gonococci, tubercle bacilli and the bacillus coli communis.

Tubercular pericarditis may arise in several ways. Thus the pericardium may be attacked in common with the other serous membranes, such as the pleuræ or peritoneum, or it may become involved as part of the manifestation of a general tuberculosis.

Again, the implication of the pericardium sometimes depends on the extension of tubercular disease affecting the pleuræ or neighbouring lymphatic glands. Pericarditis due to cancer arises in a similar way. It occasionally happens that inflammation of the pericardium occurs in the absence of any ascertainable cause, and in such instances it is termed "idiopathic." In a few cases pericarditis has been attributed to exposure to cold and prolonged exertion.

PATHOLOGY AND MORBID ANATOMY

The morbid process commences with an acute hyperæmia of the pericardial vessels, accompanied by increased exudation. The membrane becomes swollen, owing to infiltration by inflammatory effusion, and its surface appears injected, dull, and somewhat roughened by reason of the deposition upon it of fibrinous material. The inflammatory process may not advance beyond this stage (pericarditis sicca), but more commonly it goes on to the effusion of a variable quantity of fluid into the pericardial sac, from which fibrin is precipitated on to the opposed surfaces of the membrane.

The fibrinous deposit is sometimes localized, but, as a rule, it tends to spread generally, in consequence of the movements of the heart. It appears in the form of layers of yellowish lymph, which, in the early stages of the inflammatory process, is but loosely applied to the subjacent membrane, and may often be stripped off in concentric laminæ, leaving, on removal, a smooth surface.

Owing to the rubbing together of the opposed sides of the membrane, caused by the movements of the heart, the two lymph-lined pericardial surfaces become remarkably roughened, and present a variety of appearances which are often described as papilliform, shaggy, honeycombed, etc.

The amount of fibrin deposited and of fluid effused is subject to considerable variation.

The effusion may be sero-fibrinous or purulent, and is sometimes blood-stained. In rheumatic pericarditis it is almost invariably sero-fibrinous. The fluid is not infrequently stained with blood in tubercular and cancerous cases, and this is ordinarily the case when, as rarely happens, pericarditis complicates scurvy or other blood disorders, such as purpura, etc.

Purulent effusion is usually associated with pyæmic conditions, but it may be observed in connection with Bright's disease. Purulent effusion is more common in children than in adults.

Sero-fibrinous effusions tend sooner or later to become absorbed, and the visceral and parietal layers of the pericardium come again

into contact and temporarily adhere. This temporary adhesion between the two layers of the pericardium may become permanent by virtue of the absorption and organization of the fibrinous deposit on the opposed surfaces of the membrane. The attachments thus formed may be partial or complete. In the first event a general or local thickening of the membrane with a few scattered adhesions between the parietal and visceral layers may be the sole expression of a past pericarditis, while in the second the two opposed surfaces become universally adherent, and the pericardial cavity is obliterated.

In extreme cases of this kind the heart appears enveloped in a thick fibrous covering, which in a few instances has undergone a further calcareous transformation.

When the effusion is purulent the inflammatory changes are more obvious, and the pericardial sac becomes filled with a yellowish pus, which sometimes contains micro-organisms (see p. 116).

In rare instances a purulent effusion dries up into one or more caseous masses, which become intersected by fibrous adhesions, and may ultimately be absorbed or converted into calcareous material. Such a termination is, however, exceptional, since these cases usually end fatally.

Pericarditis is almost invariably accompanied by a certain degree of myocarditis, in consequence of the direct extension of the inflammatory process to the walls of the heart, the outermost layers of which become softened, and undergo fatty changes. The parts thus affected may ultimately atrophy, and be replaced by fibrous tissue. Apart from extension, myocarditis, frequently accompanies pericarditis, the two affections being the result of a common cause. In rheumatic cases the pericardium, myocardium, and endocardium, are not infrequently simultaneously involved in inflammatory changes. a condition of the heart which may be appropriately described, after Sturges, as "carditis."

The inflammatory changes in pericarditis, may also extend to the outer or fibrous coat of the pericardium, and involve the pleuræ and mediastinal tissues (pericarditis externa). By this means fibrous adhesions may be formed between the pericardium and sternum, and along the course of the great vessels at the base of the heart.

THE EFFECTS OF PERICARDITIS ON THE HEART, GREAT VESSELS, AND SURROUNDING STRUCTURES

Accumulation of fluid in the pericardial cavity must necessarily interfere with the free movement of the heart, and this statement applies especially to the diastolic expansion of the various chambers, which is seriously impeded when the effusion is considerable. The supply of blood to the organ may be impeded in consequence of pressure on the superior vena cava and left innominate vein.

A very large effusion may give rise to displacement and com-

pression of the left lung in the neighbourhood of the heart, and in addition to pressure effects on the trachea, œsophagus, and recurrent laryngeal nerves.

Dilatation of the heart, more especially of its right side, frequently follows the myocardial inflammation associated with pericarditis, and is due to the relative insufficiency of the weakened cardiac walls. The process may be perpetuated, and is no doubt sometimes initiated, by pericardial thickening and adhesion, which operate by hampering the movements of the heart, and thus increasing its work, and also by inducing muscular atrophy through pressure on the myocardium.

At the same time pericardial adhesion, even when extensive, does not invariably give rise to enlargement of the heart.

Pericardial adhesion never leads directly to cardiac hypertrophy, but it may do so indirectly by producing dilatation of the organ.

SYMPTOMS

The symptoms of pericarditis vary greatly in different cases. As a rule they are not very well marked or distinctive, and at times, as in renal pericarditis, they may be altogether absent. It frequently happens, too, that when inflammation of the pericardium occurs in association with acute disorders, such as pneumonia or rheumatic fever, the symptoms are masked by those of the primary disease.

The onset of pericarditis is shown in some instances by a general exacerbation of the symptoms of the previously existing disorder. Occasionally inflammation of the pericardium commences with a rigor, which is followed by a variable degree of pyrexia. At other times the disease begins insidiously, and, unless repeated examination of the heart be made, may be entirely overlooked.

Pain, which may be very severe, is usually an early and prominent symptom, and is felt in the præcordial and epigastric regions, and may radiate widely over the thorax and upper extremities.

Tenderness is sometimes complained of in the same situations. There is commonly a feeling of tightness or oppression at the chest, accompanied by cough and shortness of breath, which becomes more pronounced as effusion takes place.

Palpitation may be a source of great distress to the patient.

All the symptoms of fever are usually present. The temperature is raised, the tongue furred; there is thirst, loss of appetite, and the bowels are generally constipated. The urine is scanty, high coloured, and may throw down an abundant deposit of urates. It sometimes contains albumen.

As the accumulation of fluid in the pericardial sac increases, many of these symptoms are intensified. The breathing becomes increasingly difficult and laboured, so that the patient has to be propped up in bed, though in some instances of extensive effusion the decubitus is dorsal, since any attempt to raise the head tends to

increase the cerebral anæmia, and hence to cause faintness. The shortness of breath may become so great that the patient can hardly speak, and with the dyspnœa there is often a short, hacking, troublesome cough. Paroxysmal attacks of dyspnœa are occasionally observed.

The face is ashy pale, the features drawn and anxious, and the movements of the alæ nasi plainly visible. The trunk is usually kept perfectly still, while the head and arms are moved restlessly about.

With extensive effusions pressure effects on neighbouring structures may be observed.

Pain or difficulty in swallowing is due to pressure upon the œsophagus, though it has occasionally been ascribed to nerve irritation.

Irritation of the recurrent laryngeal or phrenic nerves from pressure, or from implication in the inflammatory process, may give rise to aphonia or hiccough.

Compression and collapse of the base of the left lung are occasionally observed, and are a source of additional respiratory distress.

The return of blood to the heart may be impeded by pressure on the great venous trunks in the thorax, and the veins in the neck are not infrequently distended from this cause. Thrombosis has been known to occur in the left innominate vein in cases of pericardial effusion, and is an event that would add greatly to the congestion of the veins of the head, neck, and upper extremities.

Dropsy is sometimes observed, and may be due to venous stasis or to myocardial weakness, or, as more commonly happens, to a combination of these conditions.

Cerebral symptoms in the form of headache, sleeplessness, and slight delirium are not uncommon.

Vomiting, said to be of nervous origin, is sometimes a prominent feature of acute pericarditis.

Serious delirium, convulsions, coma, etc., have been noted, chiefly in rheumatic cases, but they are of rare occurrence.

The symptoms of pericarditis in children are even more indefinite than in adults. The onset of the disease is often very insidious in young people. An increase in the pulse rate, and an irregular and excited action of the heart in association with dyspnœa, slight fever, and restlessness should always lead to a suspicion of pericarditis in the case of a child with a rheumatic history. Indeed, in any event, the occurrence of these symptoms during infancy and early life calls for a careful examination of the heart, which should be repeated from day to day.

Acute pericarditis runs a very variable course. The ordinary duration of the disease is from ten days to a fortnight, but it occasionally terminates in three or four days, and not uncommonly lasts for three weeks or more. It sometimes happens that an acute

onset is succeeded by sub-acute inflammatory changes, which run an insidious and protracted course.

Acute pericarditis usually terminates in recovery so far as the immediate issue of the disease is concerned, but in a large number of instances the heart is permanently crippled, to a greater or less extent, by the organic changes which are left behind.

Recovery in cases of slight or moderate intensity may be rapid and complete, but if there has been much effusion absorption is usually slow, and for some time after the attack there is a tendency to dyspnoea, palpitation, and other signs of cardiac weakness, when any exertion is undertaken.

Death, when it occurs, may be due to heart failure, asphyxia, or exhaustion, or to some complication of the disease with which the pericardial inflammation is associated.

PHYSICAL SIGNS

Pulse.—In the early stages of the disease the pulse is increased in frequency up to one hundred or more beats per minute, and may or may not be irregular. As a rule the artery can be felt between the beats, and is easily compressible. Later the pulse becomes extremely rapid, small, feeble, and irregular in force and frequency. In exceptional instances the occurrence of pericarditis produces very little alteration in the character and rate of the pulse.

A large pericardial effusion occasionally gives rise to the *pulsus paradoxus*.

Heart.—The earliest indications of the roughening of the opposed pericardial surfaces are obtained by means of palpation and auscultation, and consist of the signs known as “pericardial friction fremitus” and “pericardial friction sound.”

Pericardial friction fremitus is comparatively seldom observed. It is most commonly detected over the base of the heart, but it may be felt at the apex and in this situation only. Its area of production is usually small, and frequently changes from day to day. It conveys the impression that it is produced by the rubbing together of roughened surfaces immediately beneath the hand. Pericardial friction fremitus is commonly systolic as regards its relation to the cardiac cycle, but it may have a to-and-fro rhythm, *i.e.* systolic and diastolic.

It differs from an endocardial thrill in that it has no definite “focus of intensity” (Sibson), nor has it the peculiar quivering quality associated with the latter sign.

Pericardial friction sound is one of the earliest, most reliable and constant indications of the initial stage of acute pericarditis. In some instances, however, owing either to the nature of the fibrinous deposit or, as is more likely, to myocardial weakness,

the sign is wanting throughout the whole course of the disease. In its absence the recognition of the onset of acute pericarditis turns on the history and symptoms of the case, and on the occurrence of an excited, irregular, and rapid action of the heart, with which may be associated prolongation of the first sound at the apex, or reduplication of the second sound at the base and evidence of a greater or less degree of cardiac dilatation. Nevertheless a diagnosis of acute pericarditis based on the indications just mentioned is always open to doubt unless confirmed by the presence of friction sound in the præcordial area, and, as stated above, this can be detected in the large majority of instances.

Pericardial friction sound may be heard over any part of the præcordium, but it is most commonly observed over the base of the heart. Its area of audibility is, with few exceptions, limited to the præcordial outline.

Pericardial friction sound has usually a to-and-fro rhythm, *i.e.* systolic and diastolic, but it may exhibit a triple rhythm, *i.e.* presystolic, systolic, and diastolic, or it may be systolic, or diastolic only.

It is associated with the movements rather than with the sounds of the heart, and the latter, though sometimes obscured, are often audible through the friction sound.

The characters of pericardial friction sound are subject to considerable variation in accordance with the conditions under which it is produced. Thus it is described as grazing, rustling, creaking, grating, scraping, etc., and it usually conveys the impression of the rubbing together of roughened surfaces situated immediately beneath the ear.

The character, rhythm, intensity, and area of audibility of pericardial friction sound frequently vary from day to day, or even from hour to hour, and as a rule are markedly modified by pressure with the stethoscope in the præcordial area, by posture, and by the respiratory movements. As effusion takes place into the pericardial sac the inflamed and roughened surfaces of the membrane become separated by fluid from below upwards, with the result that friction, and the sound produced thereby, occur at increasingly higher levels, and in the course of time may be completely abolished. Nevertheless if a friction sound is once heard over the base of the heart it is seldom (never, according to Balfour), if ever, obliterated by subsequent effusion, however large this may be.

It must not be forgotten that the disappearance of friction sound in the course of acute pericarditis is sometimes due to the occurrence of adhesion between the two layers of the pericardium without any antecedent effusion. This order of events is observed more particularly in the case of children.

The physical signs associated with pericardial effusion will now be considered.

Prominence or bulging of the præcordial region is not uncommonly due to a large pericardial effusion, and is more readily

produced in children than in adults by reason of the greater flexibility of the chest wall in early life.

The upward and forward displacement of the ribs and costal cartilages, on which the arching of the præcordium depends, is accompanied by a widening and smoothing out of the intercostal spaces. A very large effusion has been known to exaggerate the normal curvature of the dorsal portion of the spinal column (Sibson).

When the apex beat of the heart is visible it appears to be displaced upwards together with the cardiac impulse, which becomes progressively feebler and less perceptible as the effusion and myocardial debility increase.

According to some observers, an undulatory movement can sometimes be detected in the præcordial area in cases of abundant effusion, and this they ascribe to waves of fluid produced by the beating of the heart. This interpretation of the phenomenon, if it is ever seen, is disputed by others who attribute it merely to an enfeebled cardiac impulse.

A large pericardial effusion may give rise to depression of the diaphragm and liver, and by this means to prominence of the epigastrium. Paralysis of the diaphragm, which sometimes occurs in pericarditis, would be indicated by inspiratory recession of the epigastrium and costal breathing.

Interference with the return of blood to the heart is frequently shown by distension of the large venous trunks in the neck.

The position of the apex beat and the character of the cardiac impulse are better appreciated by means of palpation. The apex beat may appear to be situated in the fourth interspace or even higher, but the impulse felt in these situations is probably due to the impact of a portion of the heart above the apex against the chest wall, the apex itself being separated from the thoracic parietes by a layer of fluid. If this explanation be correct, the so-called "displacement upwards of the apex beat" is apparent rather than real. Strongly confirmatory of this view is the fact that the apex beat may sometimes be felt in its normal situation when the patient leans well forwards, a manœuvre which brings the heart nearer to the chest wall. It must not be forgotten that in children the apex beat is normally situated in the fourth interspace.

Dr. Ewart is of the opinion that an alteration in the relation between the left clavicle and the first rib, which he terms the "first rib sign," is an important indication of a large pericardial effusion. The sign consists in a raising of the clavicle whereby the upper edge of the first rib can be felt as far forwards as its sternal attachment.

An increase in the area, and an alteration in the shape of the normal cardiac dulness, are signs of the first importance in the diagnosis of pericardial effusion.

With the patient in the recumbent position the increase of dulness is first observed at the base of the heart in the neighbourhood of the great vessels, where the pericardial sac is easily distensible. In the early stages of the effusion the dulness extends upwards as high as the second or first rib on the left side, and laterally for about an inch to an inch and a half outside the left sternal edge. As the fluid increases it dilates the pericardial sac round the heart itself and gravitates towards the most dependent part of the cavity. The area of dulness now assumes a pyriform, or roughly triangular outline, with the base of the figure downwards. Laterally, in extreme instances, it may extend from the right nipple line to the left axillary line, and upwards it may reach as high as the episternal notch.

Rotch concludes, as the result of the artificial injection of fluid into the pericardium, that a pericardial effusion gravitates from the outset to the lower part of the sac, and produces dulness to the right and left of the sternum in the fifth intercostal spaces. He considers absolute dulness in the fifth right interspace to be diagnostic of pericardial effusion, but this sign has not always the significance attributed to it by Rotch.

The area of dulness varies somewhat with the position of the patient. It is greater in the erect than in the recumbent posture, since in the latter position the fluid gravitates behind the heart. For a like reason the line of dulness may be made to move further to the right or left, according as the patient lies on his right or left side.

As a rule, the line of demarcation between the dulness due to a pericardial effusion and the surrounding pulmonary resonance is exceedingly well defined by an abrupt change in the percussion note, but it must be borne in mind that the presence of air-containing lung in front of the pericardium, such as may obtain in cases of emphysema, can seriously obscure the percussion dulness.

It occasionally happens that even a large pericardial effusion gives rise to very little increase in the area of cardiac dulness, and in such instances the fluid lies for the most part behind the heart.

The relation of the apex beat to the left limit of cardiac dulness should be carefully explored, for in many cases of pericardial effusion the area of dulness extends considerably further to the left than the apex beat.

The fingers experience a feeling of increased resistance over a pericardial effusion, and it is frequently possible, by employing the method of procedure advocated by Maguire, to map out the outline of the distended sac with the greatest nicety by means of palpation alone.

It was originally pointed out by Bamberger, and his observations have since been confirmed by others, that a considerable pericardial effusion may give rise to a well-defined area of percussion dulness,

about the size of a five-shilling piece, in the immediate neighbourhood of the angle of the left scapula. Auscultation over this patch of dulness discovers increased vocal fremitus, tubular breathing, and bronchophony.

The auscultatory evidence of pericardial effusion is limited to the effect it produces on the sounds of the heart, which tend to become weaker and more distant as the fluid increases in amount, and the myocardial debility becomes more pronounced.

The enfeeblement of the sounds is usually more noticeable at the apex than over the base of the heart, where the second sound is not uncommonly reduplicated.

A very large effusion may render the sounds altogether inaudible over any part of the præcordium.

The pressure effects³ of a pericardial effusion on the surrounding pulmonary tissue may be evidenced by the signs of partial consolidation or collapse of the lungs, more particularly of the left one over its lower lobe posteriorly.

During the process of absorption of an inflammatory pericardial effusion, the sounds of the heart become more distinct, the area of præcordial dulness gradually diminishes, and friction sound, and possibly friction fremitus, temporarily reappear as the surfaces of the membrane again come into contact.

The heart, in some instances, returns to its normal size and position, but more commonly the organ remains permanently enlarged, in consequence of the formation of adhesions, or of the effects of endo- or myocarditis.

DIAGNOSIS

If pleural and pleuro-pericardial causes can be excluded, the detection of friction sound over any part of the præcordial area justifies a diagnosis of pericarditis, while the intensity of the process may be gauged by the concomitant symptoms and physical signs.

The chief points in the differential diagnosis of pericardial, pleural, and pleuro-pericardial friction sound have already been indicated (see pp. 67, 68).

Even when friction sound cannot be heard, the occurrence of acute pericarditis may still be recognised by a careful survey of the history, symptoms, and physical signs presented by the patient.

In the absence of friction sound the diagnosis of pericardial effusion from dilatation of the heart, thoracic aneurism, or other mediastinal tumours, and pleural effusion, has also to be considered.

Dilatation of the heart may closely simulate a pericardial effusion, and a combination of the two conditions, which is by no means uncommon, renders a differential diagnosis almost impossible.

The shape of the cardiac dulness in dilatation of the heart is usually oval, whereas in pericardial effusion it is pyriform or triangular. Moreover, the upward extension of the cardiac dulness, which is so marked a feature of pericardial effusion, seldom rises

above the level of the third rib in cases of dilatation of the heart. Furthermore, in dilatation of the heart the left lower limit of dulness coincides with the apex beat, while in a pericardial effusion it frequently extends beyond this point. Rotch considers that absolute dulness in the fifth right intercostal space close to the sternum can be caused by pericardial effusion only, but, as previously pointed out, this sign has not always the significance attributed to it by this observer.

The ætiology, rate of development, and symptoms of the two conditions also furnish a means of distinguishing between them. Thus pericardial effusion commonly occurs in the course of an attack of rheumatism; it develops quickly, and is associated with pyrexia and other signs of febrile disturbance. Dilatation of the heart, on the other hand, usually accompanies valvular disease, or myocardial affections; as a rule it develops slowly (though it may do so rapidly), and in the absence of complications does not give rise to fever. It must, however, be carefully borne in mind that a greater or less degree of cardiac dilatation accompanies the large majority of cases of pericarditis. Indeed, it is to the myocardial involvement that the chief dangers of pericarditis are attributable.

The differential diagnosis between thoracic aneurism, or a mediastinal growth, and pericardial effusion can generally be made by a careful consideration of the ætiological conditions, the rate of development, physical signs, and pressure effects which are found in association with the morbid process that obtains.

The pressure effects of a pericardial effusion are seldom very pronounced.

A large pleural effusion on the left side may give rise to prominence of the chest wall in front of the heart, and thus stimulate a pericardial effusion; but the differential diagnosis does not usually present any serious difficulty unless the two conditions are combined, when it is sometimes almost impossible to distinguish between them, except by the help of paracentesis.

The presence of pus in the pericardial sac may be suspected when pericarditis complicates pyæmia, or one of the eruptive fevers, or when it follows suppuration in adjacent structures. The occurrence of rigors and elevation of temperature, which may gradually assume a hectic type, and of other grave constitutional symptoms, would convert the suspicion into a certainty. The physical signs are not distinctive of this condition, though œdema of the integuments in the præcordial region is more common in cases of purulent than of simple effusion.

PROGNOSIS

The prognosis in acute pericarditis depends mainly on the nature of the primary disorder, and on the extent to which the myocardium is involved in the inflammatory process. In the absence of previous myocardial or endocardial mischief, the prognosis in rheumatic cases is distinctly good. If the valves of the heart have been injured by a previous attack of rheumatism, the outlook is less favourable.

Pericarditis, secondary to Bright's disease, pneumonia, or pyæmia, is almost invariably fatal.

Apart from these considerations, the signs of unfavourable import are a large effusion, a rapid and irregular pulse with a feeble action of the heart, great dyspnœa, lividity, and nervous phenomena, such as muscular tremor, delirium, etc.

Persistent vomiting in the course of pericarditis in children is of grave significance, and the presence of anæmia renders the prognosis less favourable. Pericarditis is at all times a serious disorder in young people, not only on account of its immediate effects on the heart, but also by reason of the tendency of the disease to recur or continue in a sub-acute form.

TREATMENT

The first consideration in the treatment of acute pericarditis is the maintenance of complete rest in bed. Sudden movement or exertion of any kind must be carefully avoided. The patient should be warmly clad and protected from draughts, and may, if necessary, be propped up in bed by means of suitably arranged pillows.

Light and easily digestible food should be given in small quantities at short and regular intervals.

The medicinal treatment of acute pericarditis depends in a large measure on the nature of the disorder with which it is associated. It is important, where possible, to treat the primary disorder of which the pericarditis is but a local manifestation. Thus rheumatism, the commonest cause of the affection under consideration, should, when present, be combated by the usual remedies, and a similar plan of treatment must be adopted in the case of the other disorders with which pericarditis may be associated.

Local therapeutic measures are, however, of the utmost value, both as a means of relieving pain and distress and of mitigating the severity and duration of the inflammatory process. With this object the application of an ice-bag over the præcordium usually affords the most satisfactory results; but it is a method of treatment which must be employed with caution, especially in weakly subjects, and any signs of cardiac failure should be met by the administration of digitalis and alcoholic stimulants. The ice-bag also acts as a febrifuge.

Pain may also be assuaged by the use of warm poultices placed over the heart, or by the hypodermic injection of morphia.

Blisters are sometimes of service, but they have the disadvantage of rendering the physical examination of the heart more difficult.

At the onset of the disease a few leeches applied over the præcordium may give great relief, but they should be used in robust individuals only.

Whatever plan of local treatment be adopted, it ought, as a general rule, to be adhered to throughout the course of the disease, as the indiscriminate variation of the different local applications can only do harm.

The least indication of cardiac weakness should be met by the use of digitalis and alcoholic stimulants, which must be administered at regular intervals, and in such quantities as are suitable to the requirements of the case. The tendency to heart failure is more pronounced in children than in adults, and it is advisable to anticipate rather than to wait for this untoward result by the judicious use of cardiac sedatives and tonics.

One of the most useful drugs in the treatment of pericarditis is opium. It relieves pain, quiets the heart, and promotes sleep, and in suitable doses may be given to children, though alcohol is to be preferred in the case of very young subjects, in whom its action is similar (Cheadle).

Venesection is seldom required in pericarditis, but in robust individuals, with the signs of circulatory embarrassment and engorgement of the right heart, the abstraction of a few ounces of blood from the arm sometimes gives great relief.

The absorption of the inflammatory products may be hastened by the application of a few flying blisters over the præcordium, or by the internal administration of the iodide of potassium or sodium, though the action of these remedies for the purpose in question is somewhat uncertain.

When the amount of fluid effused is very large, or when the presence of pus is suspected, the question of paracentesis has to be considered.

The operation should be preceded by an exploratory puncture with a hypodermic syringe in the fifth intercostal space on the left side, about an inch from the sternum, in order to confirm the diagnosis. An aspirating needle is then introduced, under anti-septic precautions, in the same situation, and the fluid slowly withdrawn.

Rotch suggests, as the result of his experiments on the cadaver, that the puncture should be made in the fifth right space, and this method of procedure certainly seems worthy of a trial.

The operation of paracentesis pericardii is attended with little or no danger, but care must be taken not to puncture the auricles.

The pericardial cavity has in a few instances been opened by free incision and drained, a procedure which is essential for the successful treatment of a purulent effusion.

SECTION II

PERICARDIAL ADHESION

ÆTIOLOGY AND MORBID ANATOMY

Adhesion between the two layers of the pericardium, or between the pericardium and chest wall, is always of inflammatory origin. It may be the outcome of an acute or chronic pericarditis, but inasmuch as a sub-acute or chronic stage of inflammation frequently follows the acute process, the two causes are not uncommonly combined.

The distribution, extent, and characters of the adhesions, which are commonly associated with a greater or less degree of pericardial thickening, are subject to considerable variation.

Thus the parietal layer of the pericardium may be adherent to the visceral reflection of the membrane and heart (endopericardial adhesion), or to the posterior surface of the præcordial portion of the chest wall and adjacent structures (exopericardial adhesion); or the two conditions may be, and frequently are, combined. Exopericardial adhesion, involving the great vessels at the base of the heart, may arise also in connection with inflammatory affections of the mediastinum (pericarditis externa, mediastino-pericarditis, etc.), pleuræ, or contiguous organs. The adhesions, whether endopericardial or exopericardial, may be local or general. They appear, at one time, in the form of scattered filamentous threads or strands, easily torn through; while, at another, they are found as strong fibrous bands or cords, which are with difficulty separable from the underlying muscular tissue of the heart, or from the posterior surface of the sternum, as the case may be.

When the myocardial inflammation associated with pericarditis has been very extensive, the heart may appear completely encased in a dense fibrous covering, which sometimes, though rarely, undergoes a calcareous transformation.

The white opaque patches, commonly termed milk spots, which are not infrequently observed on the visceral surface of the pericardium, more particularly over the anterior surface of the right ventricle, consist of a local thickening of the serous coat of the membrane, and are due, in all probability, to inflammatory changes excited by friction.

EFFECTS ON THE HEART

The effects of pericardial adhesion on the heart are found to vary considerably. In about two-thirds of the cases of uncomplicated pericardial adhesion the heart is enlarged (Sibson), while in the remaining third the size of the organ is either unaltered or diminished.

The cardiac enlargement is usually a combination of hypertrophy and dilatation, but the latter condition commonly preponderates and sometimes exists alone.

Though the whole heart is, as a rule, implicated to a greater or less extent in these changes, the ventricles are affected much more seriously than the auricles, and the right side of the heart suffers more than the left.

The effects of pericardial adhesion on the heart are usually explained on the supposition that the fibrous attachments hamper the cardiac movements, especially systole, and thus increase the work of the organ, while its contractile power is diminished by the concomitant myocardial changes.

The influence of the adhesions in the production of cardiac enlargement has probably been over-estimated, since the pericardium may be universally adherent without giving rise to any alteration in the size of the heart. Moreover, the cardiac changes associated with adherent pericardium are probably explicable on other grounds.

It has already been pointed out that the myocardium is usually more or less implicated in the preliminary inflammatory changes, the effect of which is to diminish the amount of healthy cardiac muscle, and thus to render the heart less capable of carrying on the work of the circulation. By this means a variable degree of dilatation of the heart from failure is induced, the intensity of the process depending on the extent of the myocardial disability. Hypertrophy ensues as a consequence of the dilatation, but since the amount of myocardial tissue capable of taking part in this process is diminished, to a greater or less extent, the increase in thickness of the cardiac walls is frequently inconsiderable, and may appear insignificant as compared with that evoked by an endocardial lesion. The subsequent formation of adhesions gives rise, no doubt, to further cardiac embarrassment, and must therefore be regarded as an additional source of enlargement of the heart.

The comparative thinness of the walls of the right ventricle largely accounts for the liability of this chamber to suffer more from the effects of pericardial adhesion than the left ventricle.

In early life the formation of pericardial adhesions may greatly interfere with the subsequent growth and development of the heart, and in this way may be a cause of atrophy of the organ.

In addition to the changes mentioned above, the myocardium is not uncommonly the seat of pigmentary, fatty, or fibroid degeneration, which may be the outcome of the initial myocarditis or of the pressure exerted by the thickened and adherent pericardium on the heart itself, or on the coronary vessels.

External pericardial adhesions may involve the great arterial and venous trunks at the base of the heart, and sometimes give rise to compression and narrowing of these vessels, with the result that the free passage of blood through them may be interfered with.

SYMPTOMS AND PHYSICAL SIGNS

Pericardial adhesion is frequently unattended by symptoms of any kind. Moreover, the symptoms that may arise in connection with this condition are seldom of much diagnostic value, inasmuch as their occurrence can, as a rule, be quite as readily accounted for on the ground of functional disturbance of the heart or of some other organic cardiac lesion.

At the same time, the appearance of such symptoms as præcordial uneasiness or pain, palpitation, and dyspnoea on very slight provocation would, in the absence of a more obvious cause, suggest the presence of pericardial adhesion.

The symptoms and signs of failure of the right ventricle afford valuable evidence of pericardial adhesion, provided the other conditions which may give rise to this event can be excluded, or can be shown to be inadequate for the production of the cardiac disability.

Adherent pericardium is occasionally a cause of rapid or even sudden death.

PHYSICAL SIGNS

Although it is frequently possible to recognise the existence of pericardial adhesion by means of physical examination, it not uncommonly happens that signs are either undiscoverable or inconclusive. They are most marked when the adhesions are extensive, dense, and external, as well as internal.

The physical signs of pericardial adhesion are variously combined, but for descriptive purposes they will be considered in the order in which they would come under observation during a clinical examination of the chest and cardio-vascular system.

The Signs elicited by Inspection and Palpation.

1. In rare instances a permanent depression or flattening of the præcordial area is observed.

More commonly, however, there is bulging of this portion of the chest wall owing to enlargement of the heart from valvular disease or other cause acting concurrently with the pericardial adhesion.

2. A visible recession or retraction of the intercostal spaces, ribs, cartilages, or sternum in the præcordial area, or of certain other portions of the chest wall, coincident with the systole of the ventricles, affords valuable evidence of the existence of adherent pericardium. Systolic depression of the intercostal spaces in the immediate neighbourhood of the apex beat may occur independently of pericardial adhesion, but a visible recession of the costal cartilages, ribs, or lower end of the sternum, with the ventricular contraction, is almost conclusive evidence of this condition.

Systolic retraction of the posterior or lateral portions of the chest wall, along the line of origin of the diaphragm from the cartilages of the false ribs, is mentioned by Dr. John Broadbent as an important and reliable indication of adherent pericardium.

Diastolic collapse of the jugular veins at the root of the neck, with or without systolic retraction of the soft parts in the same situation, is occasionally observed in cases of adherent pericardium, but is of little diagnostic value.

3. The presence of diastolic retraction of the intercostal spaces in the præcordial area is of considerable diagnostic importance, since it is a sign that could hardly be caused by anything but pericardial adhesion.

4. Complete arrest of the slight respiratory movements which are normally seen in the upper part of the epigastric triangle formed by the divergence of the costal cartilages is, according to Sir William Broadbent, a constant indication of the adhesion of the heart to the pericardium and central tendon of the diaphragm.

5. Fixation of the apex beat affords valuable evidence of pericardial adhesion. The apex beat, which may or may not be displaced, does not descend during inspiration, and the position of the cardiac impulse is unaffected by change of posture.

6. On rare occasions a diastolic rebound of some portion of the chest wall in the præcordial area succeeds the systolic recession of the ribs and sternum that was mentioned above. It can be felt by the hand as an impulse during the diastole of the heart. The presence of this sign would *per se* be sufficient to establish a diagnosis of adherent pericardium.

The undulatory movements which may be seen in the præcordial area in cases of pericardial adhesion are of little or no diagnostic value.

Duroziez maintains the importance of comparing the signs afforded by inspection and palpation in the diagnosis of adherent pericardium, inasmuch as they are apparently contradictory: for during the systole of the heart the eye perceives a recession of the chest wall, while the hand detects an impulse.

The Signs elicited by Percussion

A permanent increase in the area of the superficial cardiac dulness is frequently associated with the formation of external pericardial adhesions, by reason of the attendant collapse and retraction of the superjacent pulmonic tissue, and the consequent abnormal exposure of the heart and great vessels.

Furthermore, the area of superficial cardiac dulness may be unaffected by inspiration, owing to the fixing of the anterior margins of the lungs by the adhesions.

These signs are, however, also observed in connection with inflammatory conditions of the pleuræ, hence, unless supported by other evidence, they are of little value in the diagnosis of adherent pericardium.

Evidence of enlargement of the right side of the heart, in the shape of an increase of dulness to the right of the sternum, is not uncommonly obtained.

The Signs elicited by Auscultation

Auscultation does not afford much assistance in the diagnosis of pericardial adhesion. The sounds of the heart have in some instances a peculiarly superficial character, and the second sound at the base is very commonly reduplicated. It cannot be said, however, that either of these signs is of any real diagnostic importance.

The Signs elicited by an Examination of the Pulse

The presence of adhesions between the pericardium, chest wall, and great vessels at the base of the heart was supposed to be characterized by the *pulsus paradoxus*, a condition in which the pulse wave is diminished or lost during each inspiration. It was thought that the tightening of the adhesions round the aorta, produced by the upward and outward movement of the chest wall during inspiration, led to so great a constriction of the lumen of the vessel that the heart was unable to drive blood through it; hence the pulse could not reach the wrist.

It is improbable that the lumen of a strong-walled vessel like the aorta could be constricted in the manner suggested, and, apart from this difficulty, it was observed that the sign in question was not only frequently absent under the conditions specified, but could be detected with various other lesions such as pericardial effusion, dilatation of the heart, etc., in which the mechanism of production, as described above, could not possibly obtain. For these reasons the sign has been abandoned as a distinctive indication of the presence of pericardial adhesion.

It is probable that the occurrence of the *pulsus paradoxus* depends in all cases on the inability of a weakened left ventricle to adequately meet the additional work imposed on it by the increased negative tension which obtains in the thorax during inspiration.

Consequently, it happens that during inspiration the force of the ventricular contraction becomes expended before the pulse wave reaches the wrist.

DIAGNOSIS

Post-mortem records bear ample testimony to the difficulties presented by the diagnosis of adherent pericardium. Nevertheless it is frequently possible, by means of a careful survey and comparison of the symptoms and physical signs, to recognize with certainty the existence of pericardial adhesion during life.

A history of pericarditis, or of cardiac inflammation during child-

hood, would strongly suggest the presence of adhesions, but in a large number of instances no such history is obtainable.

The diagnostic value of the various symptoms and physical signs which may be observed in connection with adherent pericardium has already been indicated, and it is necessary only to add that the information derived from these sources should be carefully weighed and compared.

At one time the symptoms, at another the physical signs, supply the evidence on which the diagnosis is based, while in a third set of cases it is some apparent inconsistency in the relation between the symptoms and signs that provides the clue to the recognition of the lesion.

The appearance of the symptoms of cardiac failure, or the presence of the signs of enlargement of the heart, would, in the absence of a discoverable or sufficient cause, suggest the existence of pericardial adhesion.

Furthermore, should the symptoms associated with a lesion of the heart be more severe than the attendant physical signs can account for, or should rupture of compensation, in case of valvular or other cardiac disease, take place without sufficient or apparent cause, the presence of adherent pericardium may reasonably be suspected.

The absence of any response to treatment may be due to a similar cause.

It has been pointed out by Sir Samuel Wilks that the occurrence of severe cardiac symptoms in young people, without evidence of valvular disease, suggests the presence of pericardial adhesions.

PROGNOSIS

If the pathological effects of pericardial adhesion on the heart are produced in the manner previously described, it follows that the prognosis of the lesion depends mainly on the extent to which the myocardium is implicated in the preliminary inflammatory changes.

If, therefore, after an attack of pericarditis the size of the heart remains unchanged, or is but slightly increased, the outlook, speaking generally, is favourable.

On the other hand, the occurrence of great enlargement of the heart, more especially in the form of dilatation, renders the prognosis much more serious.

It is probable that pericardial adhesions, unless very dense and extensive, are, *per se*, of little practical importance.

Pericardial adhesion complicated by valvular disease seldom admits of a favourable prognosis, inasmuch as the myocardial inflammatory changes usually associated with the former lesion militate against the establishment and maintenance of compensation.

TREATMENT

It is not possible to remove the organic changes associated with pericardial adhesion, hence the object of treatment is to minimize and delay, so far as possible, their injurious effect on the heart.

The nutrition of the myocardium must be promoted and maintained by suitable hygienic, dietetic, and medicinal means, and all sources of cardiac strain should be carefully avoided.

A more detailed description of the measures by which these indications are carried out will be given under the account of the treatment of affections of the myocardium.

The treatment of cardiac failure should be conducted on general principles, but care must be observed in the use of cardiac tonics.

SECTION III

HYDROPERICARDIUM

Dropsy of the pericardium is due either to (*a*) general or (*b*) local causes.

Under the first head the hydropericardium is usually one of the phenomena of general dropsy consequent on disease of the heart, kidneys, or lungs, but it may appear, in common with effusion into the other large serous sacs, during the early stages of acute Bright's disease, or in the course of the cachexia associated with tuberculosis, cancer, and the graver forms of anæmia, etc.

In rare instances hydropericardium depends on a local obstruction to the circulation through the pericardial and cardiac veins caused by thrombosis of these vessels, or by pressure upon them from without, etc. Dropsy of the pericardium is rarely large in amount. It gives rise to the signs which have already been considered in connection with pericarditis with effusion.

Hydropericardium may, however, be distinguished from pericarditis with effusion by (1) the history of one or other of the conditions mentioned above, (2) the absence of fever, (3) the absence of friction signs, and (4) the presence of general œdema and of effusion into the other large serous cavities.

The treatment of hydropericardium is that of the primary disease. A large effusion may necessitate paracentesis of the pericardium.

SECTION IV

HÆMOPERICARDIUM

Hæmorrhage into the pericardial cavity is usually due to the bursting of an aneurism of the first part of the aorta, or of one of the coronary arteries, or to rupture of the wall of the heart, the result of traumatism, or disease of the myocardium.

It may also occur, to a much slighter extent, in pericarditis due to tubercle, cancer, or scurvy, and it occasionally depends on the rupture of recent pericardial adhesions.

The clinical phenomena associated with this condition depend, to some extent, on the rapidity of the bleeding into the pericardial sac. If the hæmorrhage takes place gradually, præcordial pain, urgent dyspnœa, syncopal attacks, collapse, and evidence of loss of blood may usher in the fatal termination. A physical examination of the chest, when it can be made, discovers the signs of fluid in the pericardial cavity. The sudden discharge of a large quantity of blood into the pericardial sac usually leads to sudden death.

Treatment can be palliative only.

SECTION V

PNEUMOPERICARDIUM

Pneumopericardium, as a clinical phenomenon, is an exceedingly rare condition.

The means by which air or gas gains access to the pericardial sac is either a wound of the thorax involving the parietal layer of the pericardium, or the establishment of a communication between the pericardium and an air or gas-containing organ, such as the œsophagus, lungs, or pleuræ, stomach or intestines. In the second and more common event the extension to the pericardium of suppurative or ulcerative processes affecting one or other of these viscera is the determining cause of the perforation of the membrane.

Pericarditis quickly follows the entrance of air or gas into the pericardial cavity, and since the effusion which accompanies the inflammatory process is almost invariably purulent, a condition of pyopneumopericardium, rarely of hydropneumopericardium is produced.

The spontaneous development of gas in the pericardial cavity, consequent on the presence of gas forming bacilli, is postulated by some observers.

The symptoms associated with pneumopericardium are practically those of purulent pericarditis.

The physical signs are often very remarkable and are characteristic of the presence of gas and fluid together in the pericardial sac.

Bulging of the præcordial area is sometimes very pronounced. The hand placed over the heart occasionally experiences a succession of small shocks or vibrations due to the bursting of air bubbles at the surface of the fluid. In the recumbent posture a clear tympanitic percussion note is obtained over the cardiac area, and when the opening into the pericardial sac is patent, a cracked pot sound may be elicited.

If the patient is made to sit up and lean forwards, the tympanitic note is replaced over the lower portion of the præcordial area by a dull sound, in consequence of the displacement of the gas by fluid, under the influence of gravity.

For a similar reason a partial replacement of the lateral extent of the area of resonance by dullness can be effected by turning the patient to one side or the other.

The sounds of the heart have a characteristic metallic quality, and are described as splashing, gurgling, churning, etc. They have also been compared to the sound produced by a water-wheel in motion.

An amphoric echo of the heart sounds, and of pericardial friction sound, etc., has been observed in some instances.

In the presence of the physical signs just mentioned the diagnosis of pneumopericardium presents no difficulty. The prognosis is very unfavourable, but recovery has been recorded in cases due to injury.

There is little scope for treatment, which should be conducted on the lines indicated under the head of "Acute Pericarditis."

The question of operative procedure, in the shape of paracentesis pericardii, or of free drainage of the pericardial sac, has to be taken into consideration in every case.

SECTION VI NEW GROWTHS

The more important new growths which may affect the pericardium are tubercle, carcinoma, and sarcoma.

Tubercular disease of the pericardium is uncommon, and its occurrence is usually secondary to tubercular disease elsewhere.

A primary form of pericardial tuberculosis associated only with cessation of the bronchial or anterior mediastinal glands is described by Osler.

In a large number of instances the implication of the pericardium is due to the direct extension of tubercular disease from the lungs. The pericardium is very rarely affected in cases of general acute miliary tuberculosis.

Carcinoma of the pericardium is very rare and always secondary. The pericardium usually becomes involved by the direct extension of the growth from neighbouring structures.

The sarcomata are nearly always secondary, and are of the spindle or round cell variety.

Lymphosarcomata commonly originate in the mediastinal tissue and attack the parietal layer of the pericardium almost exclusively.

Syphilitic affections of the pericardium are almost invariably secondary to lesions of the myocardium. New growths of the pericardium usually give rise to chronic inflammation of the membrane, attended, in many instances, by effusion of blood or pus.

Clinically, the implication of the pericardium in a new growth may be suspected when the symptoms and signs of pericarditis are observed in conjunction with tubercular disease of the lungs or other organs, or with an intra-thoracic tumour.

The prognosis is, of course, hopeless, and treatment can be palliative only.

CHAPTER VIII

ACUTE ENDOCARDITIS

Classification—Section I. Acute Simple Endocarditis—Section II. Malignant or Infective Endocarditis.

INFLAMMATION of the endocardium may be either acute or chronic, and in the large majority of cases the morbid process is limited to the valves of the heart and their tendinous attachments.

It is customary to distinguish two kinds of acute endocarditis, viz. :—

1. Acute simple endocarditis
2. Acute malignant or infective endocarditis

This division of the subject is not altogether satisfactory, either from a clinical or pathological point of view, but it is the most suitable for descriptive purposes, and will therefore be adopted.

SECTION I

ACUTE SIMPLE ENDOCARDITIS

ÆTIOLOGY

Acute simple endocarditis arises most commonly in the course of an attack of acute or sub-acute rheumatism, and may precede, accompany, or follow the affection of the joints. It occurs in about 50 per cent. of the cases of acute rheumatism, and the liability of the endocardium to be affected increases with repeated attacks of rheumatic fever.

Endocarditis of rheumatic origin occurs more frequently in children than in adults, and the vulnerability of the endocardium appears to be most marked between the ages of four and twelve. The large majority of the cases of rheumatic endocarditis occur in persons under thirty years of age, and women suffer rather more frequently than men.

There is no relation between the occurrence of endocardial inflammation in acute rheumatism and the amount of pyrexia, or the severity of the joint implication.

The endocarditis which arises in the course of a considerable number of cases of chorea is, probably, of rheumatic origin.

Acute endocarditis is also observed in connection with the acute zymotic fevers, more especially with scarlet fever and measles. It occasionally arises in association with enteric fever, variola, diphtheria, and with other septic conditions, such as erysipelas, puerperal fever, septicæmia, and pyæmia.

In some instances it has occurred as a complication of pneumonia, syphilis, and gonorrhœa.

Among other occasional causes of endocarditis may be mentioned such disorders as gout, acute nephritis, and diabetes.

Acute and chronic tuberculosis are sometimes accompanied by endocardial inflammation, and in a few instances the tubercle bacillus has been demonstrated in the affected parts.

Several cases have been recorded in which endocarditis has followed a blow on the chest. Here the endocardial inflammation depends in all probability on the rupture or tearing of a valve.

The occurrence of acute simple endocarditis as a primary or idiopathic affection is mentioned by some observers, but it must be an exceedingly rare event. The exclusion of a rheumatic origin must be very difficult in cases of this kind, since it is probable that endocarditis is, in some instances, the sole expression of the rheumatic state. The presence of old-standing valvular disease, the result of acute or chronic inflammation, or of degenerative changes, is a powerful predisposing cause of acute endocarditis.

Endocarditis may be hereditary in so far as the diseases with which it is associated are hereditary. Unhealthy hygienic surroundings, poverty, and exposure are of influence in the causation of endocardial disease, by reason of the fact that such conditions predispose to rheumatism and other disorders which may give rise to endocarditis.

PATHOLOGY AND MORBID ANATOMY

The inflammatory changes in acute endocarditis are usually limited to the valves of the heart and their tendinous attachments, by reason of the greater strain to which these structures are subjected as compared with the rest of the endocardium.

Furthermore, the morbid process in adults is almost invariably confined to the valvular apparatus of the left heart. This is accounted for on the grounds that the endocardium is rendered more vulnerable by the higher blood pressure and greater variation of intra-cardiac tension which obtain on this side of the organ. For a similar reason foetal endocarditis most commonly

affects the right side of the heart. Acute endocarditis of the tricuspid valve does, however, occur in adults, and Byrom Bramwell considers that it is more common than is generally supposed. The pulmonic valve is very rarely affected.

The mitral valve is more frequently the seat of acute rheumatic endocarditis than the aortic, owing, it is supposed, to the fact that, under normal conditions, the mitral curtains are subjected to greater pressure and stress during the closing of the orifice than is the case with the aortic cusps. It is instructive to note in this connection the more common implication of the aortic valve in persons who are engaged in laborious occupations; that is, under conditions which expose the valve to strain.

The earliest and most intense inflammatory changes are observed in those portions of the valve which are most exposed to friction and pressure. These conditions are determined by the direction of the blood stream and by the lines of maximum contact of the opposed valvular segments. Consequently the auricular surface of the mitral and tricuspid, and the ventricular surface of the semilunar valves, just within the free margin of each flap, are the parts chiefly attacked.

The chordæ tendineæ are frequently implicated in the morbid process, and sometimes the general lining of the heart (mural endocarditis), especially in the neighbourhood of the valves, is attacked.

The earliest manifestation of acute endocarditis visible to the naked eye is a milky opacity of the endocardium, accompanied by the development of small translucent nodulated swellings, which resemble a string of fine beads, arranged just within the free margins of the affected valve segments.

These projections, which are the result of the accumulation in the sub-endothelial tissues of inflammatory products, subsequently increase in size, in consequence of the deposition upon them of a varying quantity of fibrin from the blood. The outgrowths, thus formed, are known as vegetations. Similar appearances may be observed in the endocardium covering the chordæ tendineæ, or other parts of the internal surface of the heart.

The vegetations are usually sessile, less commonly pedunculated, and they vary greatly in size, in accordance with the stage, extent, and severity of the inflammatory process and the amount of the fibrinous deposit.

Before describing the microscopical appearances in acute endocarditis, a brief reference must be made to the normal histological structure of the endocardium. The membrane consists internally of a single layer of flat polygonal endothelial cells, resting on a thin elastic lamina, which is supported by a stratum of nucleated branched connective tissue cells, containing a dense network of fine elastic fibres. Outside this is a layer of coarse fibro-elastic tissue, arranged in the form of trabeculæ, which becomes continuous

with the sub-endocardial connective tissue, and through this with the intermuscular connective tissue. The blood vessels supplying the endocardium are situated in the sub-endocardial connective tissue.

They accompany the muscular fibres and are not found elsewhere.

The flaps of the cardiac valves are made up of folds of the endocardium, separated and held together by fine fibro-elastic tissue.

In adults the auriculo-ventricular curtains contain striped muscular fibres in the inner thirds of their radial extent, which are continuous with the muscle of the auricular wall. The semilunar cusps are devoid of muscular tissue. It follows from what has already been said with regard to the blood supply of the endocardium, that the semilunar cusps, and the outer two-thirds of the auriculo-ventricular segments, are destitute of blood vessels, though they contain a rich network of lymphatics.

Under the microscope, the most striking feature of the morbid process, in the early stages of acute endocarditis, is seen to be a general infiltration of the structure of the valve with small round cells. They are mainly derived from the proliferation of the connective tissue corpuscles, situated immediately beneath the endothelium, though to some extent, no doubt, they are the result of a leucocytic infiltration of the inflamed part. In other portions of the endocardium the increase of the cellular elements is always most marked in the sub-endothelial connective tissue layer. The deeper layers of the membrane and the adjacent intermuscular septa are also affected, but in a less degree.

As the inflammatory process progresses the outline of the white fibres becomes indistinct, and is finally lost, so that the cells appear to lie in a homogeneous matrix. This phenomenon is exceptionally well seen in the course of the valvular inflammation, and is apparently due to the gradual absorption of the white fibres.

The cellular elements are not evenly distributed throughout the valve, but are aggregated at certain points, *i.e.* just inside the free edges of the cusps, and correspond with the small projections previously mentioned. The endothelium in these situations becomes stretched, undergoes granular degeneration, and finally desquamates, and the underlying elastic lamina also breaks up.

The small cell-capped projections are thus brought into direct contact with the blood stream, and fibrin is deposited upon them in the form of fibrillar or wavy granular masses, inclosing leucocytes, and in some instances certain micro-organisms, to wit, staphylococci, streptococci, or diplococci.

The vegetations formed in this way continue to increase in size owing to the further deposition of fibrin upon them, and they frequently acquire a villous or branched appearance by the formation of secondary outgrowths from their free extremities.

It will be observed that the vegetations are, at first, without blood vessels.

The subsequent changes consist in the gradual absorption and cicatrization of the inflammatory products, and during this process the vegetations become vascularized by the ingrowth of new vessels from the base of the valve.

The ultimate effects of an acute inflammation of the endocardium on the valves of the heart are numerous, and they vary considerably in different cases.

It is said that in its early stages the inflammatory exudation may be completely absorbed without producing any permanent alteration in the structure of the affected valve, but such a result must be exceedingly rare.

In the large majority of cases the process of healing is accompanied by fibrous thickening, and induration of the orifices and curtains of the valves.

The subsequent contraction of the newly formed tissue may give rise either to the narrowing of an orifice, or to the puckering and distortion of the segments of a valve, and thereby to its incompetence, or, as most commonly happens, to a combination of these two conditions.

The curtains of a valve sometimes become adherent to one another, or to the wall of the heart, with the production, in the first event, of valvular obstruction, and, in the second, of valvular incompetence.

The vegetations not uncommonly undergo ulcerative changes, which may lead to aneurism, perforation, or rupture of the cusps of a valve.

Rupture of the chordæ tendineæ is occasionally produced in this way, and it not infrequently happens that the efficient action of these structures is impaired by implication of the muscoli papillares in the inflammatory process.

Large vegetations may mechanically interfere with the closure of a valve, or may obstruct the blood flow through its orifice.

Portions of the vegetations sometimes become detached and swept into the blood stream, and in this way give rise to embolism in distant organs, such as the brain, spleen, kidneys, etc.

The thickened endocardium is liable, in course of time, to undergo atheromatous changes, which may be accompanied by ulceration or calcification of the affected tissues (see "Chronic Endocarditis," pp. 160).

Extension of the inflammatory process from the cardiac valves to the wall of the heart is not uncommon, and is followed by more or less intermuscular fibrosis and induration of the myocardium. The muscle fibres may undergo degenerative changes, due either to myocarditis or to compression.

Inflammation affecting the aortic valve occasionally spreads to the wall of the aorta, and may give rise to weakening and dilatation of the root of this vessel.

The secondary effects of endocarditis are the production of hyper-

trophy and dilatation of the heart, together with the changes to which these conditions are liable to give rise.

Endocarditis is frequently complicated by pericarditis or myocarditis, and occasionally by both these affections; hence the morbid appearances characteristic of one or other of these diseases may also be observed.

SYMPTOMS

The onset of acute endocarditis is usually insidious, and in the absence of complications (*i.e.* of myocarditis or pericarditis) is seldom accompanied by any symptoms that would draw attention to the heart. Moreover, the symptoms which may arise in connection with an acute inflammation of the endocardium are, as a rule, overshadowed by those of the disease with which the endocarditis is associated.

In some instances the onset of the affection is apparently shown by an exacerbation of the pre-existent febrile phenomena, by præcordial uneasiness or pain, palpitation, dyspnoea, and increased rapidity of the pulse. It is probable, however, that the occurrence of these symptoms in the course of acute endocarditis is due either to the extension of the inflammatory process to the wall of the heart, or to the incidence of pericarditis or of myocarditis. It occasionally happens that symptoms indicative of embolism are the earliest manifestation of acute endocarditis. More rarely still, the first indication of inflammation of the endocardium is the appearance of the symptoms and signs of embarrassment of the pulmonic or systemic circulation. Under these circumstances, however, the endocarditis is complicated by myo- or pericarditis, or by pre-existent endocardial disease.

The symptoms of acute or sub-acute endocarditis in children are even more obscure than in adults, and unless great watchfulness is observed the occurrence of the disease in early life may be entirely overlooked. The presence during childhood of any of the disorders which may be complicated by endocarditis, and especially the occurrence of any of the manifestations of the rheumatic state—such as arthritis, sub-cutaneous nodules, erythema, tonsillitis, etc., call for careful and repeated examinations of the heart.

PHYSICAL SIGNS

The presence of acute endocarditis can sometimes be recognised with certainty by means of a physical examination of the heart, but it not infrequently happens that the evidence afforded by this method of investigation is far from conclusive. The information supplied by inspection, palpation, and percussion is seldom of any diagnostic value.

The most reliable signs of acute simple endocarditis are elicited by means of auscultation, and consist in an alteration in the character of the sounds of the heart, or in the presence of a cardiac bruit.

The earliest and most common modification of the normal auscultatory phenomena is a prolongation and muffling of the first sound of the heart. This is most distinctly heard at the apex, and is in all probability due to impairment of the valvular element of the first sound by reason of the swollen condition of the segments of the mitral valve, which is most frequently the seat of the inflammatory process.

Prolongation of the first sound becomes audible, as a rule, during the first few days of the rheumatic attack, and may persist until convalescence is established, or it may gradually develop into an apical systolic murmur. The bruit usually possesses a soft blowing character, and is conveyed outwards into the axilla, and backwards as far as the angle of the left scapula. It indicates insufficiency of the mitral valve.

The appearance of an apical systolic murmur during the course of acute rheumatism may depend also on muscular incompetence of the mitral valve, due either to rheumatic myocarditis, or to muscular weakness consequent on the pyrexia, or the accompanying anæmia. The clinical differentiation of the cause of the murmur will be considered under the head of "Diagnosis."

A systolic murmur audible in the aortic area may be due either to a valvulitis, or to anæmia; in the pulmonic area it is almost invariably the result of anæmia.

A systolic murmur in the tricuspid area would, in the absence of evidence of previous disease of the heart, have the same significance with regard to the condition of the tricuspid valve as an apical systolic murmur has in respect to the mitral valve.

The presence of a diastolic or presystolic murmur, which is, however, not commonly heard at this stage of the endocardial inflammation, is conclusive evidence of valvular disease of the heart.

Reduplication of the first or second sound of the heart is sometimes observed as an early indication of acute endocarditis.

Reduplication of the second sound is occasionally audible at the apex and not at the base of the heart, and the second element of the reduplicated sound may be followed by a soft diastolic murmur. This form of reduplication has been insisted upon by Cheadle as an important sign of endocarditis, and he regards it as the earliest indication of commencing obstruction at the mitral opening.

The manner in which reduplication of the sounds of the heart is brought about has been considered elsewhere (see pp. 51, 52).

COMPLICATIONS

It has already been pointed out that acute simple endocarditis is commonly associated with a variable, though usually slight degree of myocarditis. An extensive lesion of the myocardium may be followed by acute dilatation of the heart.

Pericarditis complicates fully a third of the cases of acute endo-

carditis. It sometimes precedes, but more often arises simultaneously with, or follows the endocardial affection.

Rupture of the cusp of a valve, or of some of the chordæ tendinæ, is an occasional complication of acute simple endocarditis.

Acute aortitis, due to the extension of inflammation from the aortic valve, is of very rare occurrence.

Embolism and hæmorrhagic infarction of the brain, spleen, kidneys, and myocardium, etc., may occur at any stage of the endocardial inflammation, in consequence of the detachment of portions of the vegetations, or of thrombi, formed in the auricular cavities.

Pulmonary congestion, pleurisy, and pneumonia are sometimes observed in association with acute endocarditis, more especially with extensive lesions of the mitral valve.

COURSE AND TERMINATIONS

The duration and course of acute simple endocarditis are most uncertain. In a very small proportion of the cases the inflammatory process clears up without giving rise to any permanent alteration in the structure of the affected valve. It is possible, too, that in a few instances the organic changes occasioned in the valvular segments are insufficient to interfere with the functional efficiency of the valve.

In the large majority of cases, however, acute simple endocarditis leads, sooner or later, to chronic valvular disease.

Death, when it takes place during the acute stage of simple endocarditis, is usually due to the occurrence of some complication, such as acute dilatation of the heart, pericarditis with effusion, embolism, pneumonia, etc.

DIAGNOSIS

The diagnosis of acute simple endocarditis rests, for the most part, on the data afforded by a physical examination of the heart. The evidence derived from this source is, however, not uncommonly equivocal, so that the recognition of the occurrence of the lesion, although at times quite easy, is frequently difficult, and occasionally impossible. In the absence of a murmur over the præcordial area, the occurrence of acute endocarditis can be suspected only when, in the course of acute rheumatism or other predisposing cause, the symptoms of cardiac excitement or irritation make their appearance, and at the same time the first sound of the heart is observed to become prolonged.

If a murmur is heard over the cardiac region, it may be produced either inside or outside the heart.

The means of distinguishing between an endocardial and exocardial murmur have already been indicated (see pp. 70, 71).

If the presence of an endocardial murmur can be established, the differential diagnosis of its cause still remains to be considered. It has already been pointed out that an apical systolic murmur, indicative of mitral regurgitation, may be due either to inflammation of the valvular segments, or to muscular incompetence of the valve, consequent on myocarditis, pyrexia, or anæmia.

The stage of the primary disease at which the murmur is first observed furnishes the most important evidence in the discrimination of its cause. In rheumatic fever the apical murmur becomes audible, as a rule, during the first few days of the attack, whereas in other febrile disorders, such as enteric fever, etc., the bruit appears in the later stages of the disease.

In all probability, therefore, the source of the murmur in the two cases is different; hence pyrexia can be excluded as the cause of the apical systolic bruit which appears during the early stages of acute rheumatism. Anæmia can be eliminated on similar grounds, since this condition does not usually develop until the later stages of rheumatic fever. Moreover, an apical systolic murmur of hæmic origin would be preceded and accompanied by a venous hum in the neck and a pulmonary systolic bruit, and these are rarely observed during the early stages of rheumatic fever, though they may develop later.

The occurrence of acute rheumatism in an anæmic subject would render the differential diagnosis of an apical systolic murmur extremely difficult, if not impossible.

It would appear, therefore, that an apical systolic bruit audible during the first few days of rheumatic fever is in all probability due either to endocarditis or myocarditis, or to a combination of these conditions. Clinically it is difficult, indeed well-nigh impossible, to decide which of these morbid processes has given rise to the valvular incompetence. The disappearance of the murmur has been held to exclude endocarditis, but it is possible that the final changes in the valve segments produced by a valvulitis might fall short of the amount necessary to give rise to valvular incompetence.

The appearance of such symptoms as dyspnœa, vertigo, delirium, etc., in association with a small, rapid, irregular pulse and the signs of cardiac dilatation, would point to the occurrence of myocarditis, but would not exclude endocarditis.

Valvular incompetence of rheumatic origin is much more commonly due to endocarditis than to myocarditis, hence the occurrence of the former lesion is always more probable than the latter. Nevertheless, whether operative or not, a greater or less degree of myocarditis is frequently associated with acute inflammation of the endocardium.

A systolic murmur audible in the aortic area may be due (so far as the present argument is concerned) either to inflammation of the aortic valve, or to anæmia. The differential diagnosis depends on the period of the rheumatic attack at which the bruit appears, and

on the presence or absence of other signs and symptoms of anæmia.

Apart from congenital causes, a systolic murmur in the pulmonic area is almost invariably functional. The determination of the cause of the tricuspid regurgitant murmur, appearing in the course of acute rheumatism, depends on considerations similar to those which have already been discussed with reference to the mitral valve.

A presystolic or diastolic bruit is always due to organic lesions of the orifices or segments of the valves.

Accentuation of the pulmonic second sound, if at all marked, points to organic disease, probably of the mitral valve.

Signs of enlargement of either side of the heart, or a history of dyspnoea, dropsy, or of a former attack of rheumatism is indicative of pre-existent cardiac disease. The presence of an old valvular lesion, however, renders a fresh inflammation of the endocardium more likely, and its occurrence would tend to aggravate the existing cardiac signs and symptoms.

The differential diagnosis of acute simple, and malignant endocarditis will be considered under the account of the latter affection.

PROGNOSIS

In attempting to forecast the issue of acute endocarditis, the first point to be taken into consideration is the severity of the disease with which the endocardial inflammation is associated. Thus acute rheumatism, chorea, the infective fevers, etc., may each give rise to symptoms that are attended with danger to life.

In the absence of pre-existent cardiac disease the prognosis of rheumatic endocarditis is favourable so far as the immediate result of the lesion is concerned, but the probable termination of the acute inflammatory process in chronic valvular disease has also to be taken into consideration.

The condition of the heart previous to the attack of endocarditis is of considerable prognostic importance.

The existence of old-standing valvular disease greatly increases the danger to life, and does so in proportion to the extent and effects of the original lesion.

The presence of cardiac or pulmonary complications, such as pericarditis and myocarditis, or pleurisy, pneumonia, etc., also adds greatly to the gravity of the outlook.

The site and effects of the endocardial inflammation form important elements in the prognosis.

Speaking generally, incompetence of the aortic valve is much more serious than insufficiency of the mitral, whereas the danger is reversed with respect to obstruction at these openings.

The estimation of the severity of the lesion and its influence

on the prognosis will be considered under the head of "Chronic Valvular Affections."

The age, sex, occupation, habits, constitutional vigour, and general condition of the patient must also be taken into account in forming a prognosis in valvular disease following acute endocarditis, with a view to the estimation of the amount of compensation that is likely to occur, and its probable duration.

These conditions will be more fully considered under the account of the prognosis of chronic valvular lesions.

The time that elapses between the subsidence of the acute process and the period at which the patient resumes active exercise will also materially affect the extent and adequacy of the compensatory changes.

TREATMENT

The treatment of acute simple endocarditis practically resolves itself into the treatment of the disease, usually acute rheumatism, on which the cardiac lesion depends.

The preventive treatment of acute inflammation of the endocardium has hitherto been attended with little success. Nevertheless the early and active treatment of the primary disorder, and the maintenance of absolute rest, both bodily and mental, have probably some influence in warding off the occurrence of acute endocarditis.

In any case the adoption of these precautions cannot be too strongly insisted upon, more especially with respect to the manifestations of the rheumatic state in childhood, for the reasons which have already been given.

Should endocarditis occur, the measures indicated above must still be rigorously carried out. Complete rest in bed must be enforced, and all sources of cardiac irritation should, so far as possible, be removed.

Light, easily digestible food, which should consist chiefly of milk, is the most suitable diet. It is better to avoid the use of alcohol unless cardiac failure supervene.

The local application to the præcordium of poultices, fomentations, or a series of flying blisters is sometimes of considerable service in relieving cardiac pain and distress.

In young plethoric subjects, suffering from præcordial pain and oppression, the practice of venesection—at one time extensively employed—has been superseded by the use of leeches, of which two or four may be placed on the chest wall over the heart.

The local application of an ice-bag is of great service in allaying cardiac pain and distress, and in diminishing the general fever, but this procedure must be employed with caution, especially in weakly individuals.

After the subsidence of the acute symptoms the præcordium

may be painted with a solution composed of equal parts of the tincture and liniment of iodine. The application should be renewed, at intervals of three or four days, for a period of several weeks.

It is, as a rule, advisable in rheumatic cases to discontinue the use of salicylate of soda when endocarditis occurs, on account of the depressing influence which this drug exerts on the heart. It may be replaced by the administration of alkalis, with which quinine and salicin can be combined, when necessary, in the form of an effervescing mixture.

Failure of the heart may be met by the use of alcohol and other cardiac stimulants; but these remedies must be employed with caution.

In some cases small doses of digitalis or strophanthus, with ether or ammonia and strychnine, are of great service in quieting the heart. In other instances of excited cardiac action the use of anodynes, such as opium or belladonna, in small doses, is required.

Complications may be treated on general principles.

Rest should be enjoined for some weeks after the subsidence of the acute symptoms, and convalescence may be hastened by the use of cardiac and general tonics. Hæmatinics, of which arsenic is the most useful, are of great service in combating the anæmia that so commonly accompanies and follows an attack of acute rheumatism.

Iodide of potassium has been given in the later stages of acute endocarditis with the object of promoting the absorption of the inflammatory products.

Exercise should be commenced cautiously and gradually increased, and care must be taken for some months to avoid sudden or violent exertion of any kind.

SECTION II

MALIGNANT OR INFECTIVE ENDOCARDITIS

ÆTIOLOGY

This comparatively rare form of endocarditis is most commonly observed in connection with septic or pyæmic conditions. The principal sources of infection are septic wounds, abscesses, puerperal septic disease, suppurative lesions of the lungs, liver, genito-urinary passages, middle ear, etc., and acute osteo-myelitis.

Malignant endocarditis of septic origin is also found in association with acute and sub-acute rheumatism, chorea, malaria, dysentery, etc., and with some of the acute infectious fevers—to wit, small-pox, diphtheria, scarlet and enteric fevers.

Several cases have been recorded in which infective endocarditis has been associated with gall stones, unaccompanied by suppuration, in the biliary channels.

Malignant endocarditis is sometimes observed as a complication of pneumonia and meningitis.

In a few instances the disease has followed traumatic rupture of the segment of a valve.

A small proportion of the recorded cases of infective endocarditis has occurred independently of any ascertainable cause.

Malignant endocarditis most commonly attacks young adults between the ages of fifteen and thirty-five years. The disease is rare after forty, but it is not infrequently observed in children. Males suffer rather more frequently than females.

According to Osler, 75 per cent. of the cases of malignant endocarditis are complicated by old-standing valvular disease, which must therefore be regarded as a powerful predisposing cause of the affection. Among other predisposing causes of the disease may be mentioned privation, exposure to wet and cold, alcoholic excesses, chronic exhaustive disease, and the like.

PATHOLOGY AND MORBID ANATOMY

Either or both sides of the heart may be affected by malignant endocarditis. In the large majority of cases, however, the morbid process is confined to the left side of the organ. Nevertheless, implication of the right heart is, as might be expected, more common than in the simple form of the disease.

The valves are the parts chiefly affected, but it not uncommonly happens that other portions of the endocardium are also involved in this variety of endocarditis.

The mitral valve is attacked rather more frequently than the aortic, and it is not unusual for both to suffer simultaneously.

The morbid appearances, so far as the heart is concerned, are characterized by a more or less luxuriant growth of vegetations on the valves, and occasionally on neighbouring parts, and by the presence of a variable though usually considerable degree of ulceration of the valvular structures and adjacent portions of the endocardium. Two main types of malignant endocarditis are sometimes distinguished, according as the vegetative or ulcerative process predominates.

The vegetations on the valves are usually very abundant, exceedingly friable, and of a dull grey colour. They present, as a rule, a characteristic granular, fungating, ragged appearance.

The vegetations are often more or less deeply ulcerated, but the loss of substance may not be very apparent owing to its replacement by greyish or greenish coloured blood clot.

The endocardium is frequently found abraded in places, and in many instances the valves and the parts adjacent to them are the seat of deep and extensive ulcerations, which have an irregular outline and an uneven greyish yellow base.

The edges of the ulcers are commonly thickened and more or less undermined, while their floors are often wholly or partially covered by blood clot.

Ulceration is not, however, invariably present. It is most commonly observed in those cases in which malignant endocarditis has attacked a valve that is already the seat of inflammatory or sclerotic changes.

The action of the ulcerative process on the valves is usually followed by the most disastrous results, among which are aneurism, perforation or rupture of the valvular segments. In some instances the cusps of a valve undergo more or less complete destruction. Ulceration, and rupture of the chordæ tendineæ, is sometimes observed, and may lead to the detachment of a valvular curtain. The flapping of the loosened segment against the wall of the heart, with each systole, may give rise to fresh centres of infection at the sites of impact.

Ulceration of the endocardium lining the cavities of the heart may result in aneurism, abscess, or even rupture of the muscular wall of the organ.

Perforation of the septum ventriculorum has been recorded in a few instances.

Ulceration at the root of the aorta, consequent on the extension of the inflammatory process from the aortic valve, may be followed by aneurism and rupture of the vessel wall.

In addition to the changes just mentioned, evidence of pre-existent valvular disease, with enlargement of the heart, is found in three-quarters of the cases of malignant endocarditis. In a few instances the heart has been the seat of a congenital malformation.

Acute pericarditis, too, sometimes complicates malignant endocarditis.

On microscopical examination the endocardial inflammatory changes resemble for the most part those seen in the simple form of the disease. In malignant endocarditis, however, micro-organisms of various kinds are always found distributed throughout the vegetations and along the floor of the ulcers to a varying depth. They are often collected into masses, and are usually most numerous at the periphery of the vegetations. The kind of micro-organism found is not the same in all cases, and a large variety of pathogenic and non-pathogenic forms have been observed at different times. The pathogenic forms which have been shown to be capable of exciting endocarditis are the *Staphylococcus pyogenes aureus*, and *albus*, the *Streptococcus pyogenes*, the *Diplococcus pneumoniae*, and a bacillus discovered by Gilbert and Lyon.

Malignant endocarditis has occasionally been ascribed to the bacilli of tuberculosis, enteric fever, and diphtheria, and to the gonococcus. The occurrence of infective endocarditis in the course of these affections is, however, usually due to septic organisms, which also commonly accompany the diseases in question. It is probable,

too, that, in some instances, the cardiac lesion depends on the combined action of both sets of micro-organisms.

Certain special forms of micro-organisms have been described in connection with malignant endocarditis, as, for instance, the *Bacillus endocarditis rugatus*, the *Bacillus endocarditis capsulatus*, and the *Bacillus endocarditis griseus* of Weichselbaum, and the *Bacillus immobilis et fœtidus* of Fränkel and Saenger. The pathological significance of these organisms is not yet understood.

In most instances only one micro-organism is found, but occasionally the co-existence of several different kinds has been observed.

It would appear, therefore, that the organisms most commonly concerned in the production of malignant endocarditis are those found in connection with septic and pyæmic conditions and with pneumonia. Pneumonic endocarditis, however, is sometimes due to a mixed infection.

Two views are held with respect to the manner in which the micro-organisms gain access to the valves. The opinion expressed by the majority of observers is that the microbes are deposited from the blood on to the free surface of the valves, and there fructify. Others consider that the organisms are carried into the blood vessels of the valves, where they form emboli, which serve as centres for the propagation of the infection to the surface. The latter view has been opposed on the ground that the valves, for the most part, are destitute of blood vessels. This objection is not good, however, in cases of chronic inflammation of the endocardium, inasmuch as, under these circumstances, the diseased valves are abundantly supplied with blood vessels. Moreover, embolic collections of micro-organisms have been seen in the vessels of the inflamed valves by several observers. It is possible, as Dreschfeld suggests, that both methods of infection of the valves may obtain.

In addition to the cardiac lesions which have been described, malignant endocarditis is associated with a variety of other morbid conditions. The most important of these is embolism, which, from both a pathological and clinical point of view, is one of the characteristic features of malignant endocarditis.

If the endocarditis affect the left side of the heart, infarction of the spleen, kidneys, heart, brain, liver, intestines, skin, and retina is of common occurrence. When the lesion is right-sided, embolism and abscess of the lungs are observed.

It occasionally happens, more especially in the vegetative form of the disease, that a large vessel such as the middle cerebral, mesenteric, brachial, or femoral artery, or even the abdominal aorta, becomes occluded.

The infarcts, in some instances, break down with the formation of local abscesses, in which micro-organisms similar to those already noticed in the valvular vegetations may often be detected.

The stomach and intestines are not infrequently the seat of

hæmorrhagic infarcts, with congestion and ulceration of the mucous membrane. Gangrene of a portion of the intestine has been observed to follow the occlusion of a large branch of the mesenteric artery.

Inflammation of the serous membranes, especially in the form of pleurisy with purulent effusion, is not uncommon.

Croupous pneumonia, usually of a septic type, may precede or follow the cardiac lesion.

Suppurative meningitis and cerebral hæmorrhage are occasionally observed.

Micro-organisms can sometimes be discovered in the circulating blood, and septic thrombi may be found in the vessels after death.

Granular degeneration of the heart, liver, and kidneys are invariably found in association with malignant endocarditis.

The primary affection may or may not be obtrusive. As a rule, however, one or other of the morbid conditions mentioned under the head of "Ætiology" is found in connection with the cardiac lesion.

SYMPTOMS

The clinical phenomena associated with malignant endocarditis vary greatly in different cases. Moreover, the symptoms due to the cardiac inflammation are frequently insignificant, and are apt to be overshadowed by those of the general infection. It is customary, from a clinical point of view, to distinguish an acute form, and a sub-acute or chronic form of the disease. It must, however, be clearly understood that many cases of an intermediate kind occur, which do not conform exactly to either of these types. Nevertheless, this division of the subject will, with the reservation just mentioned, be adopted as the most suitable for descriptive purposes.

Two varieties of the acute form of malignant endocarditis are recognised according as the symptoms assume a typhoid or septic character.

The TYPHOID variety of the disease sometimes commences suddenly with one or more rigors, followed by headache, vomiting, rise of temperature, and grave constitutional disturbance. In other instances the onset is gradual, and takes the form of slowly increasing prostration, accompanied by a variable degree of pyrexia and ill-developed cardiac signs and symptoms.

The patient quickly passes into an apathetic state, which is attended by profound prostration, profuse sweating, and general muscular tremor.

The tongue, which is furred at first, becomes dry, brown, and fissured. Sordes form on the lips and gums. The bowels become relaxed, and melæna is not uncommon. Tympanites usually appears; the spleen is invariably enlarged and tender; and the urine frequently contains albumen and sometimes blood.

The breathing is hurried and shallow, and cough may be attended by hæmorrhagic expectoration.

The fever is usually high ($103-105^{\circ}$ F.) and of a continued type, with small daily remissions. In some of the most rapidly fatal cases, however, the temperature does not rise above 100° F.

Symptoms due to embolism of the brain, spleen, kidneys, or skin may appear at any period of the attack.

Rashes of an erythematous, papular, purpuric, or even pustular character are frequently seen on the skin.

Optic neuritis and retinal hæmorrhages are not uncommon.

Meningitis, associated with headache, drowsiness, and coma, or with delirium and convulsions, is sometimes observed.

Other complications commonly found are pericarditis, pleurisy, empyema, suppurative arthritis, and jaundice.

The cardiac symptoms are usually indefinite, but occasionally the patient complains of palpitation and præcordial uneasiness or pain. In exceptional instances urgent dyspnœa, dropsy, and other evidence of mechanical derangement of the circulation is observed.

The pulse is rapid at first, and often markedly dicrotic; in the later stages of the disease it becomes small, weak, and almost imperceptible.

The sounds of the heart are usually weak, and may be accompanied by murmurs, which are commonly systolic in the mitral, and diastolic in the aortic area. A bruit is occasionally audible over the right side of the heart. The character of the murmurs frequently changes from day to day, or even while under observation.

It is sometimes possible to elicit the signs of dilatation of the heart.

The duration of the disease is usually short. Death takes place, as a rule, within fourteen days from cardiac failure, or from the effects of the general infection, or of embolism, or of some other complication. A fatal termination has been observed on the second day of the disease. In very rare instances the affection assumes a sub-acute or chronic character.

The SEPTIC or PYÆMIC form of malignant endocarditis commences suddenly with severe and repeated rigors, followed by profuse sweating. The temperature, which is usually high and extremely irregular, may be of the remittent or intermittent type of pyrexia.

The tongue is furred at the outset, but tends later on to become brown and dry. There is commonly great thirst, and more or less complete anorexia, while vomiting is often a troublesome symptom. Tympanites and diarrhœa are frequently observed, and the spleen is almost invariably enlarged. The respirations are quick and shallow, and the pulse, which is often irregular, is rapid and easily compressible.

Embolic manifestations are of common occurrence, and metastatic abscesses may appear in divers situations. The skin may show any of the rashes previously mentioned.

Meningitis, with headache, delirium, coma, etc., is an important and common complication of this form of malignant endocarditis.

Suppurative inflammation of the serous membranes and joints, pneumonia, nephritis, and jaundice are other complications which are not infrequently observed.

A physical examination of the heart seldom discovers any distinctive indication of the endocardial lesion.

The cardiac impulse is usually feeble, and there may or may not be evidence of dilatation of the heart. Systolic murmurs are frequently audible in the mitral and tricuspid areas, but they do not differ materially from the bruits which are commonly heard, in the absence of endocardial disease, during the course of many of the acute febrile disorders.

This variety of malignant endocarditis usually runs a rapid course, in that a fatal termination is seldom delayed beyond a fortnight or three weeks.

The sub-acute and chronic variety of malignant endocarditis is termed also the "cardiac" or "malarial" type by some authors. It occurs in association with old-standing valvular affections of the heart, and is the form in which malignant endocarditis most commonly appears.

The disease generally begins insidiously, but in a few instances a more or less acute onset is observed.

In the usual course of events the patient, who is already the subject of a valvular affection of the heart, complains of general malaise. He becomes anæmic, begins to waste, and develops an irregular pyrexia, which is sooner or later accompanied by the signs and symptoms of multiple embolism.

In other instances, after a longer or shorter spell of ill health, the patient is suddenly seized with a rigor, followed by sweating, which may be repeated at irregular intervals over a period of weeks or months.

Whatever be the mode of onset, the main features of this type of malignant endocarditis are the presence of an endocardial lesion, in association with pyrexia, of a very fluctuating character, and multiple embolism. A physical examination of the heart shows the existence of cardiac enlargement and the presence of a murmur in the mitral or aortic area, or in both these situations. In rare instances evidence of right-sided endocarditis is obtained.

The murmurs, whether systolic or diastolic, often develop suddenly, and they not infrequently undergo changes in character during the course of the disease.

The fever associated with this variety of malignant endocarditis sometimes assumes a remittent or intermittent type, while, in other instances, it is more or less continuous, with occasional irregular exacerbations.

The occurrence of embolism is a constant and important feature of the chronic form of malignant endocarditis.

Infarcts are consequently produced in various organs, and, in the event of the emboli possessing infective properties, suppurative inflammation of the area of infarction takes place, with the formation of metastatic abscesses.

Embolic infarction of the brain, spleen, kidneys, or skin may be respectively indicated by hemiplegia with delirium, coma and convulsions; pain and tenderness in the splenic area; hæmaturia; and purpuric rashes on the skin.

The occurrence of pain in the chest, shortness of breath, and the spitting of blood, would point to infarction of the lungs.

Embolism of the myocardium may be followed by grave cardiac symptoms.

It occasionally happens that an artery supplying a limb becomes blocked. In this event pulsation is abolished on the distal side of the plug, and the limb becomes cold and, in rare instances, gangrenous.

Embolism of the mesenteric artery, which has been observed in a few cases, is accompanied by severe pain in the abdomen, melæna, and collapse.

Inflammation of the joints, with or without suppuration, is not uncommonly observed, and is probably of embolic origin.

An ophthalmoscopic examination may show retinal hæmorrhages or optic neuritis, or both these lesions.

The serous and mucous membranes are frequently the seat of inflammatory changes, and bleeding may take place from their free surfaces.

The spleen is usually enlarged, independently of the occurrence of infarction of the organ.

In some instances the liver is also increased in size, while slight jaundice is not uncommon.

The urine, which is frequently diminished in quantity, deposits lithates on standing. It is often albuminous, and sometimes contains blood.

The more important complications are pleurisy and pneumonia; pericarditis, meningitis, cerebral hæmorrhage, and acute aneurism.

The duration of this type of malignant endocarditis varies between a few weeks and a few months. In exceptional instances the disease has lasted for more than a year. Recovery is extremely rare, but in a few cases this result has been recorded.

PHYSICAL SIGNS

The cardiac physical signs which may be found in connection with malignant endocarditis will be briefly recapitulated.

The information afforded by a physical examination of the heart in cases of this disease is, as a rule, inconclusive, and considered apart from the general condition of the patient is seldom of much diagnostic value. The presence, however, of a cardiac murmur

of doubtful nature in association with irregular pyrexia and grave constitutional symptoms renders the existence of malignant endocarditis exceedingly probable.

Mitral murmurs occurring in this form of endocarditis, in the absence of pre-existent valvular disease, are usually systolic in time, soft and blowing in character, and audible in the neighbourhood of the apex only. The sudden appearance of a bruit of this description, which alters in character from day to day, should always lead to a suspicion of malignant endocarditis in a febrile disorder of uncertain nature.

Aortic murmurs may be either systolic or diastolic, but they are seldom well developed.

In those cases in which endocarditis of a malignant type attacks a heart, already the seat of a valvular affection, a change may be observed in the character of the murmur, previously noticed, or the extension of the morbid process to another orifice may lead to the sudden development of a bruit in a new situation.

Rapid failure of the heart, with all the signs and symptoms of acute derangement of the circulation, is sometimes observed in cases of malignant endocarditis. In other instances the cardiac dilatation comes on gradually, and is associated with passive engorgement and increase in the size of the liver, general venous congestion, and dropsy.

DIAGNOSIS

The recognition of malignant endocarditis depends, as already stated, on the presence of variable, though usually indefinite, cardiac phenomena, in association with irregular pyrexia, enlargement of the spleen, and grave constitutional symptoms, which frequently assume a typhoid or pyæmic character.

The disease has to be distinguished from a variety of other disorders, chief among which are acute simple endocarditis, enteric fever, pyæmia, meningitis, pneumonia, and acute generalized tuberculosis.

The diagnosis from acute simple endocarditis is often a matter of little or no difficulty, but at other times the differentiation between the two affections may be almost impossible, since cases occur which combine features belonging to both disorders.

In the malignant form the sudden onset (typhoid and pyæmic type), rapid prostration, and irregular pyrexia are in marked contrast to the insidious development and generally mild course of the simple variety. Signs of enlargement of the spleen, the occurrence of multiple embolism, and evidence of acute nephritis are much less seldom observed in the simple than in the infective form of the disease. Moreover, simple endocarditis rarely, if ever, gives rise to typhoid or pyæmic symptoms. Furthermore, a rapid development and a changeable character of the cardiac physical signs are more distinctive of the malignant than of the benign variety of endocarditis.

A blood count is not of very great service in the differential diagnosis of acute simple and acute infective endocarditis inasmuch as a leucocytosis has been observed in both forms of the disease. Speaking generally a pronounced leucocytosis is in favour of the existence of the infective variety. Nevertheless the occurrence of septic phenomena in association with cardiac symptoms, even in the absence of leucocytosis, is indicative of infective endocarditis.

Finally, the presence of micro-organisms, usually in the form of streptococci, which are sometimes observed in the circulating blood, would be conclusive evidence in favour of malignant endocarditis.

The diagnosis of malignant endocarditis from enteric fever is often very difficult, but the insidious onset, the characteristic rise of temperature, and the more gradual prostration of enteric fever afford important evidence in the differentiation of the two diseases. Cardiac and circulatory phenomena occur earlier, and are, as a rule, better developed in malignant endocarditis than in enteric fever. Symptoms of multiple embolism, or acute nephritis, would tell in favour of the endocardial affection. Diarrhœa, tympanites, and rose spots on the abdomen render enteric fever more probable. The skin eruptions occurring in malignant endocarditis can usually be distinguished from those of enteric fever by a careful examination. The early appearance of cerebral symptoms or of eye phenomena would be greatly in favour of malignant endocarditis.

The existence of a leucocytosis negatives the presence of enteric fever. The application of the Widal serum test would clinch the diagnosis in a doubtful case.

The differential diagnosis between malignant endocarditis and pyæmia is often impossible, since, as has already been pointed out, the two affections are to all intents and purposes identical. The occurrence of multiple embolism and the absence of a primary suppurating focus would make for a diagnosis of malignant endocarditis.

The means of distinguishing between malignant endocarditis and meningitis are found in the history and mode of onset of the two affections, and in the more common occurrence of such symptoms as squint, optic neuritis, retraction of the head or abdomen, muscular spasm and paralysis in the latter disorder.

The diagnosis of malignant endocarditis from pneumonia has to be made in those cases in which inflammation of the lungs complicates pre-existent valvular disease of the heart.

The differential diagnosis rests on the whole history of the attack, and each case must be decided on its merits, though it is sometimes impossible to come to a definite conclusion.

Acute generalized tuberculosis may closely resemble malignant endocarditis.

In attempting to make a differential diagnosis, the history and mode of onset of the attack and the presence or absence of a focus of tubercular dissemination must be taken into consideration. The existence of choroidal tubercle, or the appearance of tubercle bacilli

in the sputum, would render the diagnosis easy. The signs of cardiac disease, of multiple embolism, and of acute nephritis would be greatly in favour of malignant endocarditis. Skin eruptions, too, are more common in the endocardial affection than in acute generalized tuberculosis.

PROGNOSIS

The prognosis in the acute forms of malignant endocarditis is most unfavourable.

Life is seldom prolonged much beyond a fortnight, and a fatal issue frequently takes place within a week.

The chronic variety of the disease affords a slightly less unfavourable outlook, since recovery has been recorded in a few undoubted cases of this kind.

It is possible, however, that the application of recent methods of treatment will materially reduce the mortality from malignant endocarditis.

TREATMENT

The therapeutic measures employed in septic and pyæmic conditions are equally adapted for the treatment of malignant endocarditis.

Moreover, the various precautions mentioned under the treatment of the simple form of the disease should also be adopted.

Rest, warmth, and abundance of fresh air are of the first importance. The diet should consist of concentrated liquid food, given in small quantities at frequent intervals.

Free stimulation by means of alcohol, ether, ammonia is usually required, and should be combined with the administration of digitalis, strychnine, and other cardiac or general tonics.

Antiseptic remedies in the form of quinine, salol, perchloride of mercury, the sulpho-carbolates, etc., have been extensively used, but with little or no advantage.

Medication by means of blood serums has recently been tried, and appears likely to give better results than have hitherto been obtained in the treatment of malignant endocarditis.

The kind of serum employed varies with the form of infection which obtains, but since the large majority of cases of malignant endocarditis are due to pyogenic organisms, the anti-streptococcus serum is most commonly indicated.

Several cases of malignant endocarditis successfully treated by this serum have already been published, and although the results so far obtained have not been uniformly favourable, they are sufficiently encouraging to warrant a further trial of this method of treatment.

Benefit has apparently been derived in a few cases from the hypodermic administration of pure cultures of yeast, or of yeast-nuclein, but it cannot be said that this method of treatment affords much better results than many of the other remedial measures that have been tried.

CHAPTER IX

CHRONIC ENDOCARDITIS

Pathogenesis—Morbidity—Anatomy—Effects on the Functions of the Valves—Effects on the Heart and Circulation—Effects on other organs.

CHRONIC endocarditis is commonly the sequel of an acute or sub-acute inflammation of the endocardium, but in a large number of instances the morbid process develops gradually and insidiously, and is chronic from the beginning.

Rheumatism is by far the most common cause of chronic endocarditis in persons under middle age. During and after this period of life chronic inflammation of the endocardium (which gradually merges into and becomes indistinguishable from the morbid condition known as atheroma or arterio-sclerosis) usually depends on the strain imposed on the valves and walls of the heart by long-continued or recurrent high blood pressure in the aorta. The walls of this vessel suffer in common with the heart, and in many instances are the primary seat of the inflammatory or degenerative changes. Excessive blood pressure in the aorta is most commonly due to the protracted high arterial tension that is associated with renal disease, gout, chronic poisoning by lead or alcohol, excessive eating and drinking, and the defective elimination of the waste products of metabolism, old age, and other conditions. It is also produced by muscular strain, more especially when the effort is made in a constrained position or is accompanied by fixation of the chest and closure of the glottis, as in the case of miners, colliers, navvies, wharfingers, etc. Here, although the stress is intermittent, it acts at times with great intensity, and frequently obtains over a considerable number of years.

Syphilis, chronic alcoholism, and excessive eating and drinking, etc., which are potent factors in the causation of atheroma, no doubt frequently co-operate with strain in the production of degenerative or chronic inflammatory lesions of the cardiac valves. It is probable, too, that both gout and syphilis occasionally act as direct exciting causes of endocarditis.

The influence of chlorosis in the causation of chronic valvular changes must be ascribed to the high arterial tension which is sometimes found in association with this disorder.

Mental excitement or worry, and prolonged acceleration of the

heart's action, when combined with strain, may also be mentioned as occasional causes of chronic valvular changes.

In some instances endocarditis has followed external violence in the shape of a blow or fall on the chest.

Rupture of a valve and subsequent valvulitis is occasionally produced by sudden muscular effort.

PATHOLOGY AND MORBID ANATOMY

As in the acute form of the disease, the valvular structures on the left side of the heart are the parts most commonly affected. Right-sided valvular lesions are seldom observed, except as the result of diseases of the lungs or left heart, and in some instances of foetal endocarditis, or congenital malformation. The rarity of post-natal inflammatory affections of the pulmonic and tricuspid valves has already been accounted for under the pathology of acute endocarditis.

Chronic endocarditis of the mitral valve is usually of rheumatic origin.

The aortic valve, on the other hand, suffers more commonly from the effects of strain. In any event, strain tends to perpetuate and aggravate chronic inflammatory or degenerative changes affecting either the mitral or aortic valve.

The morbid process in chronic endocarditis consists primarily in a gradual fibrosis of the valves and their attachments, which, in consequence, become thickened, hard, and rigid. The subsequent contraction of the newly formed fibrous tissue leads to a variety of issues, among which are narrowing of the valvular orifices, puckering, retraction, and distortion of the curtains of the valves, and shortening of the chordæ tendineæ.

The thickened endocardium is very liable to be attacked by atheroma, or the disease may have been originally of an atheromatous nature, and in either event the affected structures are commonly the seat of ulceration and calcification.

Ulcerative lesions lead to thinning, or even to perforation of the valvular curtains, and it sometimes happens that the flap of a valve becomes partially or wholly destroyed by the extension of this process.

In other instances the aortic orifice, less commonly the mitral opening, is converted into a calcareous ring, and the segments of the valves not infrequently undergo a similar transformation.

The aortic valve is especially liable to atheromatous changes, owing to the frequent extension of the morbid process from the root of the aorta. The disease may in like manner spread from the aortic to the mitral valve.

Acute endocarditis often attacks the sclerosed and degenerated tissue, and may give rise to vegetations which are sometimes a source of embolism.

The endocardial changes in chronic endocarditis are not necessarily confined to the cardiac valves.

Patches of thickened tissue are often found in other parts of the lining membrane of the heart, and these may become atheromatous, and in some instances ulcerate, or undergo a calcareous transformation.

EFFECTS ON THE FUNCTIONS OF THE VALVES

The effect of chronic endocarditis is to impair to a greater or less extent the functional efficiency of the valvular apparatus of the heart, and thus to interfere with the normal circulation of blood through the organ.

The valvular inefficiency may take one of three directions, *i.e.* the damaged cusps may be unable to completely close the orifice which they guard, or the orifice itself may be narrowed, or, as most commonly happens, the two conditions are combined in varying proportions.

In the first event the valve permits the backward flow of blood, and is said to be incompetent; in the second the onward flow of blood is hindered, and the term "stenosis" or obstruction is applied to the condition of the opening.

Incompetence of a valve may occur without obstruction at the orifice which it guards, as the result either of retraction or ulceration of the valvular curtains.

Obstruction at an orifice, on the other hand, is very commonly attended by some degree of incompetence of its valve, owing to the usual inability of the valvular cusps, whether damaged or not, to adequately close the narrowed opening.

EFFECTS ON THE HEART AND CIRCULATION

Organic valvular disease of the heart, whether obstructive or regurgitant, tends to produce anæmia in front and congestion behind the seat of the lesion. This result is, however, seldom very obvious at first, except in cases where incompetence of a valve is suddenly produced, owing to the establishment of compensatory changes, which temporarily, at least, restore the balance of the circulation.

Compensatory changes always entail some degree of enlargement of the heart, and the effect of any particular lesion in this respect depends on the following general considerations:—

1. The valve affected, and the nature and tendency of the morbid process which is concerned in the production of the valvular defect.
2. The suddenness and extent of the lesion.
3. The age, sex, occupation, habits, and general condition of the patient.

These points will be considered in detail under the account of the prognosis of valvular affections.

Enlargement of the left side of the heart is very commonly accompanied by hypertrophy of the right ventricle, which, by keeping up the blood pressure in the pulmonic circulation, comes to the assistance of the left ventricle.

Disease at any cardiac orifice tends in course of time to produce an increase in size of the openings situated behind the seat of the lesion. As a rule, regurgitant lesions are more effective in this respect than obstructive disease in the same situations. Indeed, according to Professor Hamilton, stenosis of the mitral or aortic opening causes little or no enlargement of the pulmonic and tricuspid orifices, but this view is opposed to the opinion of the majority of writers on diseases of the heart. Nevertheless, an increase in size of the pulmonic opening is hardly ever observed, and incompetence of the pulmonic valve is one of the rarest of cardiac lesions.

EFFECTS ON OTHER ORGANS

Valvular disease of the heart leads, sooner or later, in the large majority of cases, to congestion of the pulmonic and systemic circulations, and is, in consequence, attended by disturbance of function of the various organs throughout the body.

Moreover, long-continued venous congestion of the viscera induces organic changes which are most marked in the lungs, liver, stomach, spleen, kidneys, brain, and heart.

The pulmonic lesions most commonly found are—congestion and œdema of the lungs, dilatation and rupture of the pulmonic capillaries, pulmonary apoplexy, brown induration of the lungs, bronchitis, and lobar pneumonia.

In the early stages of valvular disease the liver becomes enlarged and congested. Atrophy and fatty degeneration of the hepatic cells ensue, and ultimately the fibrous tissue of the organ is increased. On section the viscera presents the typical “nutmeg” appearance.

The mucous membrane of the stomach and intestines is congested, and may show hæmorrhages. Catarrhal inflammatory changes are commonly observed throughout the alimentary tract.

The spleen is enlarged and firm. It shows on section a dark purple colour owing to congestion. Infarcts may be seen, and the fibrous stroma of the organ is increased.

The kidneys are usually enlarged and indurated. On section the medullary portions are seen to be engorged with blood, and there may be signs of old or recent infarction. The kidneys sometimes show cirrhotic changes.

The vessels of the brain are usually congested, and the organ itself may be shrunken. Signs of embolism may be seen.

The myocardium is not uncommonly the seat of pigmentary,

granular, fatty, and fibroid changes, with corresponding alterations in the texture of the muscular substance of the heart.

The effects of the above-mentioned changes on the functional activity of the various organs act and react on one another, so that a "vicious circle" of events becomes established, which sooner or later terminate in the death of the patient.

Thus the interference with the functions of secretion and excretion leads to the imperfect assimilation of food and the accumulation of waste products in the blood, effects which are aggravated by the impaired functional activity of the nervous system and the improper oxygenation of the blood in the lungs. Nutrition is thereby seriously interfered with, and the muscular substance of the heart suffers in common with the other tissues. The cardiac malnutrition leads to further circulatory embarrassment, which in turn aggravates the existing functional derangement of the organs throughout the body.

CHAPTER X

CHRONIC VALVULAR LESIONS

Pathogenesis of Chronic Valvular Disease.

UNDER this head it is convenient to include all the pathological processes which may lead to an abnormal condition of any portion of the valvular apparatus of the heart.

The causes, therefore, of chronic valvular lesions may be classified as follows:—

1. Acute or sub-acute endocarditis.—The lesion is usually on the left side of the heart, and the mitral valve suffers more commonly than the aortic.

Either incompetence of a valve or obstruction at an orifice may be produced, or, as usually happens, the two conditions are combined in varying proportions. The occurrence of obstruction at an orifice without incompetence of its valve is a very much rarer event than that of incompetence of a valve without obstruction at the orifice which it guards.

2. Chronic endocarditis (including atheroma).—The lesion is again most commonly situated on the left side of the heart, and in cases of rheumatic origin the mitral valve suffers more frequently than the aortic. The aortic valve is usually the seat of the chronic endocardial changes produced by strain and atheroma.

Here again obstruction at an orifice is nearly always accompanied by incompetence of its valve. Incompetence of a valve sometimes occurs without obstruction at its aperture, but in the large majority of cases the two conditions are combined.

3. Rupture of the cusp of a valve or of some of the chordæ tendineæ.—This lesion may be due to sudden muscular effort, or to external violence, and is productive of valvular incompetence.

4. Imperfect coaptation of the curtains of a valve owing to dilatation of the orifice which they guard.—This lesion gives rise to valvular incompetence. Aortic incompetence due to this cause is a very rare event, and depends on a general dilatation of the first part of the aorta, involving the orifice of the vessel.

Relative insufficiency (as the condition is usually termed) of the pulmonic valve is practically unknown.

Mitral regurgitation is not uncommonly due to dilatation of the orifice of the valve, whereby the proper adaptation of the flaps is prevented.

The enlargement of the mitral opening is, in the large majority of cases, part of a general dilatation of the left ventricle, and the muscular constriction of the orifice, which takes place during systole, is insufficient to enable the valvular curtains to come into apposition. Defective muscular closure of the valve is in some instances the sole cause of its insufficiency.

Tricuspid incompetence is, *mutatis mutandis*, brought about in a similar way.

5. Congenital malformation of the valves and intra-uterine endocarditis.—Pulmonic stenosis, which is frequently associated with some other structural or valvular defect of the heart, is the most common congenital malformation of the cardiac valves.

Intra-uterine endocarditis usually affects the right side of the heart, and the pulmonic valve suffers more frequently than the tricuspid.

CHAPTER XI

MITRAL INCOMPETENCE

Pathogenesis — Morbid anatomy — Compensation : its method of production and maintenance — Failure of compensation, and its results — Symptoms — Complications — Physical signs — Diagnosis — Estimation of the amount of regurgitation.

ÆTIOLOGICAL PATHOLOGY

UNDER normal conditions the complete apposition of the two segments of the mitral valve during the ventricular systole, whereon the closure of the opening depends, is obtained by means of (*a*) the narrowing of the aperture that is produced by the contraction of the muscular fibres surrounding it, and (*b*) the contraction of the muscoli papillares, which, through the chordæ tendineæ, draws down and approximates the curtains of the valve, and at the same time prevents their eversion into the auricle. Imperfect closure of the mitral orifice, and consequently leakage into the left auricle during the ventricular systole, may be due to any cause which interferes with the accurate apposition of the curtains of the valve. Thus it may depend on (*a*) structural disease in any portion of the valvular apparatus, (*b*) enfeeblement of the muscular structures concerned in the efficient working of the valve, and (*c*) defective co-ordination of the series of events which determine the perfect action of the valvular mechanism. As a consequence of acute or chronic endocarditis the free edges of the curtains of the valve may become thickened, retracted, ulcerated, or otherwise deformed, so that the adequate apposition of the cusps is rendered impossible. In other instances the flaps are perforated, or they become adherent to one another, or to the walls of the heart, or their proper adaptation is prevented by a growth of vegetations.

The valvular inadequacy depends, in some cases, on atheromatous changes, which most commonly attack the large anterior segment of the valve, and may lead to softening, ulceration, or calcification of this structure.

The chordæ tendineæ may be thickened, rigid, or shortened, but, on the other hand, they sometimes become stretched, and occasionally rupture. The papillary muscles frequently show degenerative changes, and in some instances the muscle fibres are more or less replaced by fibrous tissue.

The rupture of a cusp, or of one or other of the chordæ tendineæ,

or muscoli papillares, is, in occasional instances, the cause of mitral insufficiency.

Regurgitation through the mitral orifice is very commonly due to relative or muscular incompetence of the valve.

The two conditions are usually combined, and are the result of dilatation of the left ventricle from long-continued strain (such as accompanies protracted high arterial tension, aortic valvular disease, etc.), and of muscular enfeeblement, consequent on anæmia, prolonged fevers, myocarditis, and malnutrition, or degeneration of the myocardium from any cause.

The incompetence of the mitral valve that not infrequently arises during middle life and old age is due to one or other of these causes acting singly or in combination.

The valvular incompetence depends on the inability of the segments of the valve to come into complete apposition, in consequence either of the increase in the size of the mitral orifice which accompanies dilatation of the left ventricle (relative incompetence), or of the insufficient constriction of the opening by its enfeebled muscular sphincter during the ventricular systole (muscular incompetence).

It is obvious, however, that relative incompetence must necessarily be accompanied by muscular inadequacy, whereas muscular inadequacy, sufficient to give rise to valvular incompetence, might exist without producing any dilatation of the opening; but, as previously mentioned, the two conditions are almost always combined.

PATHOLOGICAL RESULTS

The Effects on the Heart and Circulation

In consequence of the incomplete shutting of the mitral valve, blood is forced backwards into the left auricle during the ventricular contraction. The auricle, therefore, now receives blood from two sources, *i.e.* from the pulmonic veins, and from the left ventricle, and at the end of its diastole its contents exceed the normal by the amount which has regurgitated from the ventricle.

The accommodation of this increased quantity of blood involves dilatation of the auricle from overfilling. The auricular walls subsequently hypertrophy, in consequence of the additional work entailed by the propulsion into the left ventricle of a greater amount of blood than usual; but owing to the nature of the lesion and the thinness of the muscular covering of the auricle, dilatation is always in excess of hypertrophy.

The abnormal strain to which the walls of the auricle are subjected usually gives rise also to more or less thickening of the endocardium lining the cavity.

Owing to the increase in the auricular contents, blood is propelled in larger quantity, and with greater force than usual, into the left

ventricle during diastole, so that dilatation of the chamber from overfilling is produced. Hypertrophy also occurs as the result of the increased work involved in the expulsion of the unusually large volume of blood contained in the ventricle and in the maintenance of the tone of the ventricular wall.

The high pressure which prevails in the left auricle, especially during the ventricular systole, is felt also in the pulmonic veins and capillaries, and gives rise to interference with the outward flow of blood in these vessels. The obstruction to the free passage of blood through the lungs, thus induced, is followed by a general rise of pressure in the pulmonic circulation by reason of the more vigorous action of the right ventricle in the face of the additional work that is required of it. Under favourable circumstances the increased functional activity of the ventricle results in hypertrophy of its walls, which is essentially a conservative process, since, by maintaining a high blood pressure in the pulmonic circuit and left auricle, it lessens the regurgitation through the mitral opening, and thereby increases the quantity of blood supplied to the aorta.

Long-continued high tension in the pulmonic circulation gives rise, sooner or later, to more or less thickening, congestion, and dilatation of the pulmonic vessels, with fibrotic induration of the lungs.

The condition known as "brown induration" of the lungs represents these changes in an advanced stage.

It will be observed that compensation in cases of mitral regurgitation is effected chiefly by means of hypertrophy of the right ventricle. The hypertrophy of the left auricle is insufficient to be of much practical service. Dilatation of the left ventricle, if combined with adequate hypertrophy, is beneficial in so far as the increased capacity of the chamber neutralizes, to some extent at least, the amount of back-flow; but the powerful systole, while augmenting the blood supply to the aorta, also aggravates the leakage through the mitral opening, and in this respect is harmful.

The development, maintenance, and duration of the compensatory changes depend on the following considerations:—

1. The quality and quantity of the blood supplied to the myocardium.
2. The suddenness, extent, and tendency of the valvular lesion.
3. The recuperative power and endurance of the patient as evidenced by his age, family history, habits, general condition, and so forth.
4. The presence or absence of complications.

Incompetence of the mitral valve, slowly developed, of moderate severity, and stationary in character, occurring in a young and otherwise healthy adult, may, in the absence of complications, give rise to compensatory changes which endure for years, and more or less completely restore the normal balance of the circulation.

In the majority of cases, however, compensation is less perfect, with the result that a diminished supply of blood to the aorta and systemic vessels leads to arterial anæmia, accompanied by congestion in the pulmonic area, and later by a varying degree of general venous plethora.

Failure of compensation is usually slowly developed, in consequence either of the progressive character of the lesion, or of the gradual enfeeblement of the muscular power of the ventricles, induced by long-continued overwork. The ordinary sequence of events characteristic of cardiac failure may, however, be interrupted at any stage of the process by the occurrence of pulmonic or other complications, which, not infrequently, give rise to acute dilatation of the heart.

Lung affections, especially bronchitis, pneumonia, pleurisy with effusion, etc., and intercurrent attacks of pericarditis or endocarditis, are a fertile source of failure of the right ventricle, owing to the additional strain thereby thrown on the already overtaxed walls of this chamber.

In whatever manner produced, failure of compensation is characterised by a series of further changes, which, unless capable of removal, terminate in the death of the patient.

Owing either to the absolute or relative weakness of its muscular walls, the right ventricle fails to completely discharge its contents at each systole, and in consequence undergoes gradual dilatation from incomplete emptying. Coincidentally with this process, muscular and relative incompetence of the tricuspid valve is established. The blood supply to the right auricle now exceeds the normal by the amount which escapes backwards, with each systole, through the tricuspid valve; consequently the chamber undergoes dilatation from overfilling. Hypertrophy of the auricular walls may ensue, but this is seldom well marked, and from anatomical considerations alone can be of little practical value. Further dilatation of the right auricle from incomplete emptying quickly supervenes, and is accompanied by congestion and engorgement of the venæ cavæ and vessels of the portal and systemic venous systems.

Moreover, the establishment of tricuspid incompetence must necessarily be followed by a fall of blood pressure in the pulmonic circulation and left auricle, the effect of which is to still further increase the leakage through the mitral orifice, and thereby add to the embarrassment of the right ventricle, in addition to diminishing the supply of blood to the aorta.

It will thus appear that failure of compensation in cases of mitral disease results in the establishment of a train of events, which act and react injuriously on one another, and tend to the production of arterial anæmia, accompanied by ever-increasing pulmonic and systemic venous engorgement.

The effects of this "back-working" process on the functional activity and structural arrangements of the heart, lungs, brain,

liver, and other abdominal organs have already (see p. 163) been described, and need not be recapitulated. Dropsy due to cardiac causes first manifests itself in those parts furthest away from the heart, where presumably the circulation is most feeble. It commences, therefore, in the feet, and gradually spreads upwards, and may ultimately involve all the subcutaneous tissues. Effusion into the serous cavities of the abdomen, pleuræ, pericardium, etc., also occurs, and collections of fluid, even when small in amount, in any of these situations are always a source of serious cardiac embarrassment.

SYMPTOMS

In all forms of chronic valvular disease of the heart the occurrence of symptoms and the severity of their manifestation are determined, for the most part, by the degree of adequacy of the compensatory changes. The more perfect the compensation, or, in other words, the more nearly the normal balance between the arterial and venous circulations is restored, the fewer and less important are the symptoms.

In some instances compensation is so complete that the patient is not aware that he is the subject of heart disease, but in the majority of cases it is less adequate, and sooner or later the symptoms and signs of cardiac insufficiency make their appearance. The period of occurrence and the character of the symptoms are by no means constant for any given lesion, and they vary greatly in different forms of valvular disease, but in all cases they are referable to mechanical disturbance of the systemic and pulmonic circulations, or to disordered action of the heart.

In fully compensated cases regurgitation through the mitral orifice may give rise to no symptoms, but as a rule patients suffering from this disease complain of shortness of breath when any unusual effort is made. The liability to dyspnoea on exertion, which is common to all forms of compensated valvular disease, is much more marked in the early stages of mitral than of aortic affections. The reason is that lesions of the mitral valve must, from the outset, lead to a rise of pressure in the pulmonic circulation, whereas aortic disease, in its early stages, has little or no influence in this respect. As compensation begins to fail the dyspnoea becomes more pronounced and more easily excited, and is often associated with cough, which is sometimes dry and irritable, while at others it is attended by a profuse watery expectoration.

Attacks of palpitation, accompanied by irregularity of the heart, and præcordial uneasiness, or pain, may be of frequent occurrence, and may arise on very slight provocation.

In some instances the patient first comes under observation complaining of dyspepsia, flatulence, and irregular action of the

bowels, or it may happen that œdema of the feet and legs is the earliest indication of the cardiac insufficiency.

In any event shortness of breath soon becomes a troublesome and prominent symptom, and is quickly followed by evidence of congestion of the lungs and other viscera.

The effects of venous engorgement of these organs are the same for all forms of valvular disease, and since they are so marked in mitral lesions, and especially in mitral regurgitation, a general account of the symptoms arising therefrom will be given in this place.

Pulmonic.—The earliest and most important indication of pulmonary congestion is dyspnœa, which gradually increases in severity as the cardiac failure becomes more pronounced. In the final stages of the disease the difficulty in breathing frequently takes the form of orthopnœa.

Paroxysmal attacks of dyspnœa of an asthmatic nature are by no means rare in the course of mitral affections.

Cough is often very troublesome, and hæmoptysis sometimes occurs in consequence of the rupture of a vessel in the lungs.

Examination of the lungs reveals the presence of œdema of the bases and dependent parts, and not uncommonly the signs of bronchitis.

Hydrothorax may occur as part of a general dropsy, or as the result of an acute inflammation of the pleural membrane.

Epistaxis is not uncommon, and may give rise to serious loss of blood.

Portal.—Enlargement of the liver, with tenderness of the organ, is one of the earliest indications of interference with the flow of blood through the right heart.

Gastro-intestinal congestion is attended by loss of appetite, nausea, dyspepsia, flatulence, and irregular action of the bowels. Hæmatemesis and melæna sometimes occur, and hæmorrhoids may be a source of great discomfort.

Genito-Urinary.—Renal congestion is shown by a diminished secretion of urine, which is high-coloured, and throws down, on cooling, a copious deposit of lithates. Albumen is usually present in small quantities. Casts are not found in the absence of structural disease of the kidneys.

Uterine congestion is accompanied by disorders of menstruation and occasionally by menorrhagia.

Cerebral.—Headache, giddiness, faintness, vertigo, and especially sleeplessness, are associated with the congestion of the circulation through the brain. Delirium sometimes occurs, and is of grave import. Cheyne-Stokes' respiration occasionally attends the final stages of failure of the heart.

Cutaneous.—The patient commonly presents an anæmic appearance, while the skin has often a dirty yellow tinge. The conjunctivæ are pale. Cyanosis of the lips, face, and extremities is frequently observed, though, on the other hand, in not a few instances a condition of extreme pallor is found in these situations.

The patient loses flesh, and this is especially noticeable in the case of children.

Dropsy begins with swelling of the ankles, and gradually spreads upwards. It may involve the serous cavities. Thrombosis in the large venous trunks may lead to a peculiarly solid form of œdema in the area drained by the vessel that is affected. The swollen subcutaneous tissues may be the seat of erysipelatous inflammation, or even of gangrene.

Hæmorrhages into the skin are not uncommon, but they are usually very small.

Failure of compensation may be recovered from, and its effects removed by means of suitable treatment, but each attack leaves the heart and tissues more or less crippled and less capable of resisting the further inroads of the disease.

As a rule death, in cases of mitral incompetence, takes place slowly, and depends on gradual enfeeblement and dilatation of the ventricles, and especially of the right, since it is on this chamber that the stress of the lesion mainly falls.

Sudden death in mitral regurgitation is of comparatively rare occurrence, but it may happen as the result of embolism or cerebral hæmorrhage.

COMPLICATIONS

The commonest and most important complications of mitral regurgitation are those affecting the lungs. Acute bronchitis, pulmonic œdema, pleurisy with effusion, pneumonia, etc., may at any period of the disease give rise to urgent cardiac symptoms by reason of the additional strain thereby imposed on the right ventricle.

Extensive dropsy and especially effusion into the pericardial, pleural, or peritoneal cavity, add greatly to the embarrassment of the heart.

Embolic manifestations may appear at any stage of the disease. The spleen, kidneys, brain, and skin are the organs most commonly affected.

Thrombosis sometimes occurs in the large venous trunks, and may give rise to various symptoms.

Severe cardiac pain is occasionally associated with mitral incompetence, but it very rarely happens that an attack of angina pectoris is experienced.

Pericarditis is not an uncommon complication of mitral disease, and is more frequently observed in children than in adults.

PHYSICAL SIGNS

Physiognomy.—The observation of the physiognomy in cases of morbus cordis forms an important part of the physical examination, and often furnishes valuable indications of the nature and severity of the lesion.

Attention should be directed mainly to the following points, viz. the expression, complexion, colour of the skin, attitude, conformation, state of nutrition, and general appearance of the patient.

The face in mitral regurgitation often appears somewhat suffused and puffy, and is frequently the seat of venous stigmata, scattered about either singly or in clusters. There is commonly more or less cyanosis of the lips, cheeks, ears, nose, and extremities, though in some instances the complexion is of an ashy grey hue. The conjunctivæ and skin, with the exception of the parts above-mentioned, are usually anæmic, and in the late stages of the disease frequently present a dirty yellow discolouration, in consequence, no doubt, of the continued congestion of the liver.

The attitude of the patient is often highly suggestive. Thus there may be orthopnoea, or the position assumed may be such that the accessory muscles of respiration can be most advantageously brought into play.

Clubbing of the fingers is sometimes observed in old-standing cases of mitral incompetence.

The muscles become soft and flabby, and loss of flesh is by no means uncommon. The emaciation may be masked by dropsy of the subcutaneous tissues, especially in the terminal stages of the disease.

Pulse.—During the period of compensation the pulse is, as a rule, fairly regular in frequency, but irregular in force. The artery is of medium size, and cannot usually be felt between the beats. The pulse wave is short, badly sustained, and easily compressible. With the appearance of symptoms the pulse rate is increased, and the beats become markedly irregular both in force and frequency. The pulse wave grows shorter, less sustained, and more easily obliterated by pressure with the finger, and the vessel is empty between the beats. Speaking generally, the pulse of mitral regurgitation is characterized by irregularity as regards both force and frequency, and also by low tension. For further information the reader is referred to the section dealing with the pulse.

HEART

Inspection.—Bulging of the præcordium is sometimes observed in young subjects, with yielding chest walls.

The apex beat of the heart is displaced outwards and slightly

downwards. The outward displacement is due partly to the dilatation of the left ventricle and partly to the enlargement of the right heart, which tends to tilt the apex outwards. The downward displacement of the apex beat is due to the hypertrophy of the left ventricle. Epigastric pulsation is often well marked, in consequence of the forcible contraction of the hypertrophied right ventricle.

Palpation.—The impulse of the heart is diffused and forcible, and is usually best appreciated over the right ventricle and in the epigastrium.

A thrill, systolic in time, can sometimes be felt at the apex.

Percussion.—The area of cardiac dulness is increased chiefly in a lateral direction, and to the right more than to the left. The reason for this is obvious. The upper limit of dulness is slightly raised in consequence of the enlargement of the left auricle.

Auscultation.—At the apex the first sound is accompanied by a blowing systolic murmur, which is transmitted outwards into the left axilla, and not infrequently backwards, as far as the angle of the left scapula.

The murmur is sometimes propagated upwards and inwards, and may be audible along the left sternal edge as high as the third or second rib. It occasionally happens that the murmur can be heard over the whole præcordium, and in exceptional instances over the whole thorax.

The murmur is usually loudest at the apex, and gradually “tails off” as the stethoscope passes into the axilla on the one hand, or towards the sternum on the other.

In rare instances the loudness of the bruit is most marked over the second left interspace and adjoining ribs, close to the sternum. The phenomenon is supposed to depend on the nearer approach than usual of the enlarged left auricle to the chest wall, in consequence of the retraction of the left lung.

The systolic murmur heard at the apex may partially or wholly obscure the first sound, or may follow it.

At the base of the heart the sounds over the aortic cartilage are unchanged, but over the pulmonic cartilage the second sound is accentuated. Reduplication of the second sound is often observed over the base of the heart. When the right ventricle fails, and tricuspid incompetence is established, the accentuation of the pulmonic second sound is lost owing to the fall of blood pressure in the lungs. A systolic murmur due to tricuspid regurgitation may become audible over the ensiform cartilage and base of the sternum. The concurrent signs of tricuspid incompetence are distension and occasional pulsation of the veins in the neck, enlargement of the liver, and congestion of the portal and systemic venous circulations, together with its effects. Hepatic pulsation is also sometimes observed.

DIAGNOSIS

The diagnosis of mitral regurgitation rests mainly on the presence of a systolic bruit at and to the left of the apex, in association with accentuation of the pulmonic second sound, and the signs of enlargement of the right ventricle. It sometimes happens that the murmur is absent, or inaudible, as, for instance, when the ventricular contractions are either very feeble or extremely rapid. Under these circumstances the diagnosis, which is seldom a matter of much doubt, must be based on the concomitant signs and symptoms of pulmonic and systemic venous engorgement, and of dilatation of the left and right sides of the heart.

A systolic murmur in the mitral area is sometimes of exocardial origin, and may depend on disease of the pericardium, pleuræ, or lungs. The differential diagnosis of exo- and endocardial bruits has already been considered (see p. 70), and seldom presents much difficulty in adults.

In children, however, a systolic exocardial murmur at the apex due to pericarditis may closely simulate the bruit of mitral regurgitation. The difficulty of differentiating between the two is increased by the fact that relative incompetence of the mitral valve not uncommonly depends on the myocarditis that so frequently accompanies pericardial inflammation.

Systolic murmurs produced at the aortic, pulmonic, and tricuspid orifices are sometimes audible at the apex, but their site of maximum intensity, and the direction of their transmission, taken in conjunction with the condition of the right and left sides of the heart, and the characters of the pulse, usually suffice to distinguish them from mitral bruits.

If these sources of error can be excluded, and the diagnosis of mitral regurgitation is thereby established, the cause of the valvular insufficiency still remains to be considered.

Regurgitation through the mitral orifice may depend on (a) organic disease of some portion of the valvular apparatus, or (b) muscular relaxation of the opening whereby the segments of the valve are unable to come into complete apposition during systole.

The differential diagnosis is a matter of considerable importance from the point of view of treatment, inasmuch as the latter condition is curable, while the former is not.

The two morbid processes sometimes co-operate in the production of mitral incompetence, and it is then practically impossible to estimate their relative influence and importance.

In practice it is found that the difficulty in distinguishing the cause of mitral insufficiency arises most commonly in connection with the appearance of an apical systolic murmur in association with anæmia, or during the course of an acute febrile disorder.

The reason for this is that while anæmia and acute febrile dis-

orders are prolific sources of muscular incompetence of the mitral valve, they are also associated, either directly or indirectly, with the conditions under which organic valvular disease may arise.

Thus the differential diagnosis of mitral incompetence which is observed in a patient who presents a definite rheumatic history, and is also the subject of well-marked anæmia, or who is suffering from acute rheumatism, scarlet fever, or measles, etc., may give rise to considerable difficulty.

The recognition of the cause of the mitral insufficiency in cases of this kind rests, for the most part, on the history and general condition of the patient, taken in conjunction with the cardiac physical signs.

For instance, in the case of a patient suffering from anæmia, the differential diagnosis of incompetence of the mitral valve depends on the following considerations:—

1. *The history and general condition of the patient.*—A history of acute rheumatism would be evidence in favour of organic disease of the mitral valve. On the other hand, well-marked pallor of the skin and mucous membranes, with no emaciation, and the absence of the signs of congestion in the pulmonic and systemic venous circulations, would point to incompetence of the valve from myocardial enfeeblement.

2. *The condition of the heart and the characters of the murmur, pulmonic second sound, and pulse.*—In anæmia the apex beat is seldom displaced to any great extent, the ventricular systole is not forcible, and the signs of cardiac enlargement, when present, are those of dilatation, and not of hypertrophy.

Generally speaking, a murmur due to muscular incompetence of the mitral valve follows, and does not replace, the first sound of the heart. Moreover, the bruit is usually soft and blowing in character; it is not well conducted, and is seldom heard in the left axilla, or at the angle of the left scapula. Furthermore, a murmur at the apex, due to anæmia, is almost invariably preceded and accompanied by a venous hum in the neck, and a pulmonic systolic bruit.

Accentuation of the pulmonic second sound is rarely well marked in the absence of organic disease.

The tension of the pulse is increased in many cases of anæmia, especially in the early stages of the disorder, and this is never observed in organic disease of the heart productive of incompetence of the mitral valve.

3. *The effects of treatment.*—Cardiac murmurs, due to anæmia, disappear under the administration of hæmatinics, so soon as the blood is restored to its normal condition, and the healthy nutrition of the cardiac muscle is re-established.

The diagnosis of the cause of the mitral insufficiency which is frequently observed during the course of an acute febrile disorder,

such as acute rheumatism, enteric fever, measles, etc., has already been considered under the head of "Acute Endocarditis" (see p. 146).

It turns on the period of the attack at which the apical systolic murmur is developed, and on the presence or absence of the signs and symptoms of mechanical disturbance of the circulation. In many instances, however, the solution of the problem is impossible until some time has elapsed after the subsidence of the acute process.

The differentiation of the cause of the incompetence of the mitral valve, which is commonly associated with chorea, is based on considerations similar to those already indicated.

In exophthalmic goitre, again, a like method of reasoning is applicable.

The muscular and relative incompetence of the mitral valve which attends dilatation of the left ventricle from long-continued strain in cases of aortic disease and protracted high arterial tension hardly come within the scope of the present discussion, and are considered elsewhere.

Prolonged high arterial tension is a cause both of organic valvular disease and of myocardial overwork, hence it plays an important rôle in the production of mitral insufficiency. In cases of this kind it is almost impossible to determine whether the valvular incompetence is due to organic disease, or to muscular enfeeblement, or to a combination of these conditions.

A similar difficulty is frequently experienced in the differential diagnosis of the mitral insufficiency which comes on insidiously during and after middle age.

THE ESTIMATION OF THE AMOUNT OF REGURGITATION IN MITRAL INCOMPETENCE

The estimation of the extent of the valvular lesion, or rather of the amount of blood which regurgitates into the left auricle during each systole, is based on the information derived from the following sources:—

1. **The characters of the pulse.**—Attention in this respect should be directed chiefly to the size and strength of the pulse and to the condition of the artery between the beats.

A small, weak and short pulse of low tension, associated with a forcible ventricular contraction, is, for obvious reasons, indicative of a considerable amount of regurgitation. On the other hand, if the artery can be felt between the beats, and the pulse is regular and of fair size, length and strength, the inference is that the amount of leakage into the auricle is slight.

2. **The degree of enlargement of the left side of the heart.**—The position of the apex beat, the force of the impulse and the outline of dulness will enable the observer to gauge fairly accurately

the size of the left auricle and ventricle, and the strength of the ventricular contraction.

Dilatation of the auricle and ventricle is, to a certain extent, a conservative process, and provided it is not excessive, and is accompanied by adequate hypertrophy, as shown by the downward displacement of the apex beat and the force of the ventricular contraction, the lesion is probably slight.

Great enlargement of the left ventricle, especially in an outward direction, is indicative of serious regurgitation.

3. The degree of enlargement of the right ventricle.—A moderate degree of hypertrophy and dilatation of the right ventricle is compatible with an inextensive mitral lesion, provided dyspnoea is not easily excited and there is an absence of the signs of pulmonic congestion.

Great increase in the size of the right side of the heart, especially when associated with shortness of breath and other evidence of pulmonic engorgement, is indicative of serious incompetence of the mitral valve.

4. The character and conduction of the murmur and its relation to the first sound of the heart.—Neither the intensity nor the quality of the systolic apical bruit affords any criterion of the severity of the lesion, though, generally speaking, a loud or long murmur denotes that the ventricle is acting vigorously, whereas a soft or short one may indicate impending failure of the heart. Nor can the manner in which the murmur is conducted be said to have much significance with respect to the estimation of the severity of the lesion. A bruit audible in the axilla and at the angle of the left scapula may accompany either slight or extensive regurgitation, but, as a rule, the greater the amount of regurgitation, the better is the murmur propagated towards the left.

It is to the effect of the murmur on the first sound of the heart that attention should be mainly directed. The more the first sound is replaced by the systolic bruit, or, in other words, the less capable the auriculo-ventricular curtains are of giving rise to the sound of tension, the more serious is the incompetence of the mitral valve.

Thus a systolic murmur heard at the apex, which *follows* and does not obscure the first sound and is not audible at the angle of the left scapula, denotes that the leakage through the mitral opening is slight.

On the other hand, if the first sound is totally obscured by the murmur, which is conducted into the axilla and is heard at the angle of the left scapula, the inference is that the amount of regurgitation is very considerable.

5. The degree of accentuation of the pulmonic second sound.—So long as the tricuspid valve is competent, the amount of accentuation of the pulmonic second sound forms one of the most reliable

indications of the degree of tension in the blood vessels of the lungs, and hence of the extent of the leakage through the mitral valve.

6. **The severity of the symptoms.**—Provided there are no complications or other extraneous exciting cause, and there is evidence on physical examination of the heart of well-developed compensatory changes, the occurrence of severe symptoms is significant of serious mitral incompetency.

To recapitulate, the signs of an inconsiderable amount of regurgitation through the mitral valve are: (*a*) a pulse of fair size, length and strength, and an artery that can be felt between the beats; (*b*) moderate enlargement of the left and right sides of the heart; (*c*) apical systolic murmur which follows and does not obscure the first sound, and is not audible at the angle of the left scapula; (*d*) slight accentuation of the pulmonic second sound; and (*e*) the absence of habitual dyspnoea, cough, etc., and of the signs of pulmonic and systemic venous congestion.

On the other hand, the signs of a large amount of leakage through the mitral opening are: (*a*) a small, weak, short, irregular pulse of low tension; (*b*) great enlargement of both sides of the heart, but more especially of the right ventricle; (*c*) an apical systolic murmur which wholly or partially obscures the first sound; (*d*) well-marked accentuation of the pulmonic second sound; and (*e*) habitual dyspnoea, and the signs of pulmonic and systemic venous congestion.

CHAPTER XII

MITRAL STENOSIS

Pathogenesis—Morbid anatomy—Pathological effects on the Heart and Circulation
—Symptoms—Complications—Physical signs—Diagnosis—Estimation of the degree of Stenosis.

ÆTIOLOGICAL PATHOLOGY

IN a large proportion of the cases, obstructive disease at the mitral orifice is the result of chronic endocarditis of rheumatic origin. Moreover, this variety of mitral disease appears to arise much more commonly in connection with the slighter manifestations of rheumatism than with the more acute forms of the disorder, a fact which goes far to explain the comparative frequency of its occurrence in childhood and early life.

Mitral stenosis has also been ascribed to congenital causes, but the weight of evidence, both pathological and clinical, is largely against this mode of origin.

In a considerable number of instances, narrowing of the mitral orifice, among adults, has been found in association with chronic renal disease and arterio-sclerosis. The relation between the valvular affection and the renal and arterial changes has not yet been satisfactorily determined.

Narrowing of the mitral aperture has been attributed to anæmia, by reason of the high systemic tension and consequent irritation to which the mitral valve is exposed in some cases of this disease.

A more likely cause of irritation of the valvular segments is the more or less constant vibration of these structures that is maintained by the to-and-fro flow of blood, which accompanies the mitral insufficiency so commonly associated with anæmia. Moreover, it is to be expected that an overgrowth of fibrous tissue in the valvular curtains and basal ring would be more readily excited in young anæmic subjects than in adults by irritation of this kind.

The more common occurrence of mitral stenosis among women than men may, in part at least, be accounted for by the fact that girls suffer from chorea and anæmia so much more frequently than boys.

The morbid process which results in narrowing of the mitral

orifice usually consists in a gradual and progressive fibrosis of the segments of the valve, chordæ tendineæ, and basal ring, whereby these structures become thickened, rigid, and contracted.

The effect of these changes on the size and shape of the opening varies greatly in different cases, but the valvular deformity that is produced can generally be included under one of two kinds, which have been termed the "funnel-shaped" and "button-hole" varieties respectively.

In the "funnel-shaped" form which is commonly found in young subjects, and sometimes in old people, the basal ring may be more or less contracted. The segments of the valve are thickened, and the opening they inclose is constricted, while the free edges of the aperture are pulled upon by shortened and rigid chordæ tendineæ, so that the appearance presented by the valve is that of a cone with its apex pointing into the ventricle.

In the "button-hole" variety, which, with few exceptions, is the condition found in adults, the basal ring is greatly thickened and rigid. The curtains of the valve are replaced to a variable extent by dense fibrous tissue, which incloses a narrow slit representing the auriculo-ventricular orifice. The chordæ tendineæ and muscoli papillares may participate in the morbid process, and are often found converted into a thick mass of fibrous tissue.

Calcic and other salts are frequently deposited in the sclerosed tissue, and in some cases the mitral orifice is transformed into a calcareous ring.

The two forms of valvular deformity may be variously combined, with corresponding modifications in the morbid appearances.

Obstruction at the mitral orifice is sometimes caused by a mass of valvular vegetations, and in rare instances narrowing of the opening has been produced by large calcareous nodules in the muscular wall of the ventricle (Byrom Bramwell).

Stenosis of the mitral opening is usually accompanied by some degree of incompetence of its valve on account of the inability of the thickened rigid and distorted curtains to adequately close the narrowed orifice.

PATHOLOGICAL RESULTS

Effect on the Heart and Circulation

The earliest effect of interference with the passage of blood through the mitral orifice is to increase the work of the left auricle, and thereby to produce hypertrophy of its walls. Furthermore, since the lesion in the large majority of cases is progressive, the additional work required of the auricle, and therefore the amount of its hypertrophy, increase *pari passu* with the degree of obstruction which obtains.

Sooner or later, however, owing to the progressive constriction of

the mitral orifice, the hypertrophied auricle becomes unable to completely discharge its contents at each systole, and consequently undergoes dilatation from incomplete emptying, that is, from failure.

The interference with the return of blood from the lungs that is occasioned by the gradual increase of pressure which takes place in the left auricle from the outset, is met by hypertrophy of the right ventricle and a general rise of tension in the pulmonic circulation.

The occurrence of dilatation of the left auricle aggravates the difficulties of the pulmonic circulation, and in this way gives rise to additional hypertrophy of the right ventricle, and a further increase of tension in the blood vessels of the lungs.

The hypertrophy of the right ventricle in mitral stenosis is greater than in any other form of valvular disease of the left side of the heart. Moreover, the congestion of the pulmonic circulation and the changes produced thereby are more pronounced than in mitral incompetence on account of the greater and more continuous strain to which the blood vessels of the lungs are exposed in mitral stenosis. Thus in mitral regurgitation the tension in the left auricle and pulmonic circulation is diminished during the ventricular diastole, whereas in mitral stenosis it persists, for the most part, throughout the whole cardiac cycle.

The abnormally high blood pressure in the lungs frequently leads to the rupture of a vessel, hence hæmoptysis is not an uncommon symptom in cases of mitral stenosis.

Auscultatory evidence of pulmonic regurgitation is occasionally observed in association with mitral stenosis. Incompetence of the pulmonic valve under these circumstances is in all probability due to interference with the valvular mechanism by the pressure of an enlarged auricular appendix, in which blood clotting may occur, and is not due to chronic forcing of the valve, else it would be found much more commonly than is the case.

In the terminal stages of mitral obstruction, dilatation of the left auricle is usually very much in excess of hypertrophy, and in some instances the enlargement of the chamber is so great that pressure is exerted on surrounding structures. Moreover, thrombosis is apt to occur in the dilated left auricle, or in its appendix, on account of the slowing of the circulation through the chamber. The clots thus formed are sometimes carried into the blood stream, and by this means give rise to the embolism of distant organs.

The left ventricle in mitral stenosis is usually found either unaltered or slightly diminished in size. This is accounted for by the fact that the ventricle receives, on the whole, a smaller quantity of blood than usual from the left auricle, and has, in consequence, less work to perform in the propulsion of its contents into the aorta. In the advanced stages of the affection the diminished supply of blood to the left ventricle, and hence to the aorta, leads to a considerable degree of arterial anæmia. Enlargement of the left ventricle (*i.e.* hypertrophy or dilatation, or a combination of the

two conditions), which is observed in about half the cases of mitral stenosis, is ascribable to an initial incompetence of the valve, or to a concurrent local cause, such as pericarditis, etc., or to an extra-cardiac affection, as, for example, chronic renal disease.

The adequacy of the compensatory changes in mitral stenosis turns on the extent to which the hypertrophied left auricle, with the help of the hypertrophied right ventricle, is able to maintain a sufficient supply of blood to the left ventricle and aorta. The rôle of the left auricle in this respect is subsidiary to that played by the right ventricle, which bears the whole brunt of the pulmonic circulatory embarrassment.

It is thus easy to understand the important influence exerted by pulmonary complications in the production of failure of the right ventricle in mitral stenosis.

Compensation may be maintained for years, and the conditions on which its duration depend have already been considered under the head of mitral regurgitation.

The breakdown of compensation in mitral stenosis may be due, in the first instance, to dilatation from failure either of the left auricle or of the right ventricle, but it commonly depends on the latter cause.

The concurrent establishment of tricuspid incompetence is accompanied by a fall of blood pressure in the lungs, and the occurrence of general venous engorgement, visceral congestion and their effects (see pp. 172, 173).

Dropsy is usually a late phenomenon in mitral stenosis, and is seldom well marked. The early occurrence of œdema may, however, be observed in those cases in which obstruction at the mitral orifice is combined with incompetence of the valve.

SYMPTOMS

The symptoms of mitral stenosis do not differ materially from those observed in mitral incompetence. It will therefore be unnecessary to do more than emphasize the features of special interest in connection with the symptoms of mitral stenosis.

It occasionally happens that the discovery of the physical signs of mitral obstruction precedes the occurrence of symptoms. With a slight or even moderate degree of stenosis compensation may be maintained for years, and during this time the patient suffers little or no discomfort of any kind.

Shortness of breath on exertion, accompanied in many instances by palpitation and a feeling of tightness or constriction of the chest, may, for considerable periods, be the only indication that the heart is working under difficulty. Pain is seldom an obtrusive feature.

Epistaxis is not infrequently observed during the early stages of mitral stenosis.

Hæmoptysis is often an early symptom, and it commonly happens

that the patient seeks advice under the impression that he is suffering from an affection of the lungs.

Dyspepsia or an attack of bronchitis is often the first warning of commencing failure of compensation. Symptoms referable to congestion of the pulmonic circulation are especially obtrusive during the course of failing compensation in cases of mitral stenosis.

Dyspnoea is always a prominent feature of the disease under these conditions, and paroxysmal attacks of difficulty in breathing are not uncommon.

Cough, attended by the expectoration of mucus, which is often blood-stained, may be a troublesome symptom.

Attacks of bronchitis are of frequent occurrence, and are a fertile source of failure of the right ventricle.

Patches of pneumonic consolidation or of pulmonary apoplexy are, also, not uncommonly observed.

The influence of pulmonary complications in the production of failure of the right ventricle is even more important in mitral stenosis than in mitral incompetence.

A type of mitral stenosis, which has been insisted on by Petit, is characterised by the presence of well-marked anæmia in association with dyspnoea, palpitation, constipation, nervous disturbances, and catamenial troubles.

Emaciation is sometimes a very prominent feature of mitral stenosis, more especially in young subjects.

Embolic manifestations are more common in mitral obstruction than in any other form of chronic valvular disease. The arteries of the brain, spleen, and kidneys are the vessels most frequently blocked. The plugging of a cerebral artery may be followed by necrosis of the area of brain supplied by the vessel. On the other hand, the function of the affected part is frequently wholly or partially restored by the establishment of a collateral circulation. Dilatation of the right ventricle from failure, as in mitral regurgitation, leads to the signs and symptoms of portal and systemic venous stagnation. Dropsy, however, is rarely well marked in mitral stenosis, and as a rule does not appear until the terminal stages of the disease. The reason for this is not fully understood. Should dropsy occur at an early stage of the disorder, the stenosis is almost invariably found to be associated with incompetence of the mitral valve. Extreme dropsy in mitral stenosis is, as Sir William Broadbent has pointed out, highly suggestive of concomitant tricuspid obstruction.

PHYSICAL SIGNS

Physiognomy.—The crimson lips and bright pink flush on either cheek, which are so often observed in cases of stenosis of the mitral orifice, form a striking contrast to the appearance presented by the subjects of incompetence of this valve.

The complexion in mitral stenosis has not, however, always these characters. In some instances the face exhibits the signs of anæmia, and is of a pale, sallow colour; while in others, and especially when the stenosis is associated with well-marked incompetence of the mitral valve, the lips and cheeks present a dusky hue, suggestive of imperfect aëration of the blood. In the terminal stages of the disorder, when failure of the right ventricle has supervened, the appearance of the patient resembles that seen in cases of mitral incompetence.

Pulse.—So long as compensation is maintained the pulse is regular, both in force and frequency. The artery is small, and can be felt between the beats, but is easily compressible. The pulse wave is small, long, and easily obliterated by pressure with the finger.

When compensation fails, and the tricuspid valve becomes incompetent, the pulse becomes extremely irregular, both in force and frequency. If stenosis of the mitral orifice is associated with incompetence of its valve, the pulse is usually irregular from the beginning.

The condition of virtual tension of the pulse, which is present in cases of mitral stenosis until a late period of the disease, is of considerable interest, and is probably the outcome of reflex vasomotor influences, whereby the systematic vessels contract down on their diminished contents. In the presence of chronic renal disease this explanation is, of course, unnecessary.

HEART

Inspection.—Bulging of the præcordial region is very rarely observed in uncomplicated cases of mitral stenosis. The apex beat is seen in its normal position, or it may be displaced slightly outwards and downwards.

Visible pulsation is occasionally manifested in young, thin subjects, in the second and third left intercostal spaces close to the sternum, and is supposed to be due to the systole of the enlarged left auricle.

The forcible contraction of the hypertrophied right ventricle frequently gives rise to epigastric pulsation.

Palpation.—The apex beat is usually ill defined, but the impulse of the left ventricle is, as a rule, fairly distinct, and communicates to the hand the sensation of a short, sharp shock, or tap.

Palpation in the region of the apex beat frequently reveals the presence of a peculiar vibratory thrill, which immediately precedes, and terminates abruptly in the ventricular impulse. Pulsation over the right ventricle is diffused, forcible, and heaving. The ventricular

impulse is felt most distinctly when the hand is placed over the epigastrium.

Percussion.—The area of cardiac dulness is increased upwards, and to the right, and in some instances to the left also. The upper limit of dulness may reach to the level of the third or even second rib, and is due to dilatation and hypertrophy of the left auricle.

When the enlargement of the right ventricle is well marked, the line of dulness may extend laterally beyond the right sternal edge.

In uncomplicated cases of mitral stenosis the left ventricle is not enlarged; in point of fact, it is often somewhat smaller than usual. Consequently there is little or no alteration in the left limit of cardiac dulness, or in the position of the apex of the heart. An increase of the cardiac dulness to the left, with displacement of the apex beat outwards and downwards, depends mainly on dilatation and hypertrophy of the left ventricle, which is most commonly due to concurrent incompetence of the mitral valve. It may also depend on other causes, as, for example, peri- or myocarditis, chronic renal disease, etc.

Auscultation.—At the apex a rough vibratory presystolic murmur is heard, which runs up to, and terminates abruptly in a short, sharp and loud first sound. The first sound, in this situation, may also be accompanied or followed by a soft blowing systolic bruit, indicative of concurrent mitral incompetence. The second sound may or may not be audible at the apex. The presence or absence of the second sound at and to the left of this point is determined by the degree of mitral narrowing which obtains.

At the base the second sound is frequently reduplicated, and a similar phenomenon is sometimes observed at the apex.

In certain instances the double sound is audible at the apex *only*, and the sign is then of considerable diagnostic significance, in so far as it is one of the earliest indications of commencing obstruction at the mitral orifice (see p. 53).

Over the pulmonic cartilage the second sound is accentuated, while in the aortic area it is either unaltered or somewhat diminished in intensity.

Several of the foregoing physical signs require more detailed consideration.

In typical cases the presystolic murmur is peculiarly rough and rumbling in character, and it increases in intensity up to its abrupt termination in the first sound.

As a rule the murmur is audible over a very limited area, which is situated just above and internal to the apex beat. It is very rarely propagated into the left axilla, nor can it be heard at the angle of the left scapula. In exceptional instances, however, a very loud bruit is audible in both these situations. It occasionally happens, too, that the murmur is conducted towards the right, when

it may be heard along the left sternal edge as high as the third rib or second interspace.

The characteristic features of the presystolic bruit, therefore, are its peculiar quality, singular intensity, and the limited area of its audibility.

It is, however, not the only kind of diastolic murmur associated with mitral stenosis.

During the period distinguished by Sir William Broadbent as the second stage of the disorder, the diastolic murmur is extremely variable and inconstant. The different modifications of the diastolic murmur which may be observed are conveniently classified as follows:—

1. The early diastolic murmur (or, as it is sometimes termed, the post-systolic murmur).
2. The mid-diastolic murmur.
3. The late diastolic, presystolic, or auriculo-systolic murmur.
4. The entire diastolic murmur.

The different varieties of the diastolic murmur may be represented diagrammatically (see opposite page).

The early diastolic bruit is of somewhat rare occurrence as an isolated murmur in mitral stenosis, but when present it closely follows the second sound, and is audible during the beginning only of the diastolic period. It is a short bruit, of variable character, and exceedingly inconstant, so that it may even appear and disappear during the time of examination.

A murmur occupying the middle portion of the diastole has been termed mid-diastolic. It resembles the foregoing bruit both in character and in the irregularity of its appearance.

The presystolic variety of the diastolic murmur has already been described and is the one most frequently heard in cases of mitral stenosis.

A bruit occupying the whole of the diastolic period is of fairly common occurrence in mitral stenosis, and is termed the entire diastolic murmur. It commences immediately after the second sound, and during the latter part of the diastole it gradually increases in roughness and intensity up to its abrupt termination in the first sound.

The murmur may be cut into two portions by an appreciable interval, so that the sequence of events is a diastolic bruit immediately following the second sound (early diastolic murmur), then a period of quiet, succeeded by a late diastolic or presystolic murmur, running up to the first sound.

The method of production of the different varieties of the diastolic murmur, and the conditions under which each one is occasioned, will now be briefly considered.

DIASTOLIC MURMURS

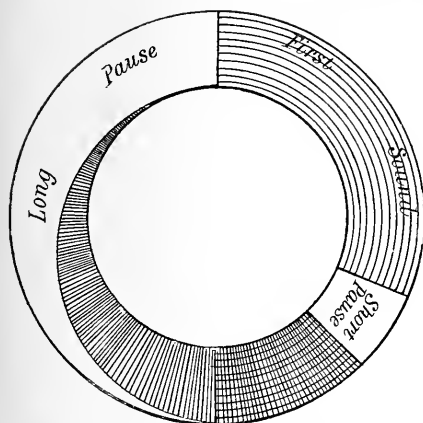


FIG. 38. DIAGRAMMATIC REPRESENTATION OF AN ENTIRE DIASTOLIC MURMUR

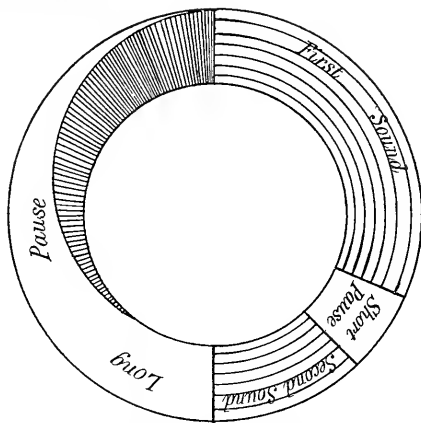


FIG. 39. DIAGRAMMATIC REPRESENTATION OF A PRESYSTOLIC MURMUR

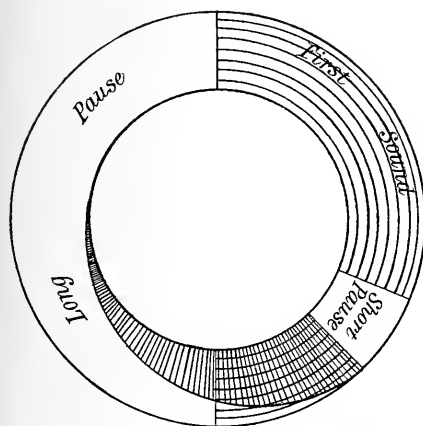


FIG. 40. DIAGRAMMATIC REPRESENTATION OF AN EARLY DIASTOLIC MURMUR

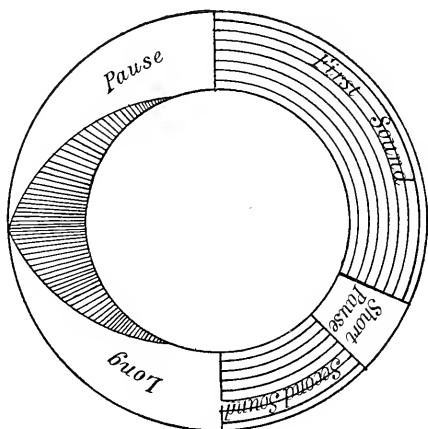


FIG. 41. DIAGRAMMATIC REPRESENTATION OF A MID-DIASTOLIC MURMUR

In compensated cases of mitral obstruction the forces concerned in the propulsion of blood through the narrowed auriculo-ventricular orifice into the left ventricle are :—

1. The high pressure in the pulmonic vessels and left auricle which is maintained by the hypertrophy of the right ventricle.
2. The force of the auricular systole.
3. The suction power of the left ventricle during diastole.

Provided these forces, either separately or collectively, are sufficiently powerful, during any period of the diastole of the heart, to bring about the requisite swiftness of flow through the narrowed mitral opening into the left ventricle, a "fluid vein" is formed. Consequently the conditions necessary for the production of a murmur are present.

The variety of diastolic bruit which is produced depends on the period, during the diastole, at which the above conditions are fulfilled.

Thus they may be present during the auricular systole only, or during the active dilatation of the left ventricle, or they may obtain during the whole diastole with the production of presystolic, early diastolic, and entire diastolic murmurs respectively.

The loudness of the murmur depends mainly on the rapidity of the blood current. It is on this account that the presystolic bruit is usually so intense, for the forces urging the blood into the ventricle are all combined during the time of the auricular systole, and this is the period during which the murmur in question is produced.

It is beyond the scope of this work to consider the objections which have been raised against these views, and it must suffice that the explanations given above are those accepted by the majority of observers.

The cause of the short, sharp first sound in mitral stenosis has been the subject of considerable discussion, and is still a matter of dispute.

It has been ascribed to the sudden and forcible contact of the mitral curtains, which, in consequence of the imperfect distension of the left ventricle, are not so gradually floated up or so perfectly adapted to each other during diastole as obtains under normal conditions.

Dr. Sansom considers that such forcible contact of the mitral segments during the ventricular systole is impossible, both on physiological and mechanical grounds. He suggests that the sound is produced on the right side of the heart, and is due to the rapid and vigorous action of the right ventricle acting in the face of considerable resistance, whereby the wall of this chamber, in conjunction with the tricuspid valvular curtains, gives rise to the "sudden sound of tension."

An objection to this view is the fact that the occurrence of failure

of the right ventricle is not followed by any alteration in the character of the first sound.

Sir William Broadbent has proposed another explanation. He supposes that in consequence of the imperfect filling of the left ventricle the wall of this chamber is suddenly "brought up" taut on its contents after a portion of the systole is completed.

The disappearance of the second sound at and to the left of the apex, which is observed when the constriction of the mitral opening is at all considerable, has been ascribed by Broadbent to (1) the weakening of the aortic second sound, consequent on the lessened diastolic tension of the aortic semilunar valves that follows a diminished supply of blood to the aorta, and (2) the displacement from the chest wall of the left ventricle, which normally conducts the aortic second sound to the apex, by the enlarged right heart.

The last stage of mitral stenosis is characterized by the absence of the presystolic murmur, so that all that is heard at the apex is a short, sharp first sound with or without a systolic bruit.

The disappearance of the presystolic murmur is due to the development of tricuspid incompetence which leads to a fall of pressure in the pulmonic circulation. The effect of this is that the auricular systole now drives blood backwards as well as forwards, and the flow into the left ventricle is not sufficiently forcible to generate a murmur. At the same time, the accentuation of the pulmonic second sound is lost, and a systolic bruit, due to tricuspid regurgitation, may become audible over the ensiform cartilage.

The concomitant signs of insufficiency of the tricuspid valve are distension of the jugular veins, enlargement of the liver, and congestion of the systemic and portal venous circulations, together with its effects.

In the terminal stages of mitral stenosis the liver may fail to enlarge on account of the supervention of cirrhosis of the organ, induced by long-continued venous congestion. This event makes the prognosis more serious, and may be a cause of persistent ascites, even when there is little or no general œdema.

Dropsy usually appears late in mitral stenosis, and is seldom well marked.

The early occurrence of dropsy in mitral obstruction will generally be found to depend on concurrent incompetence of the mitral valve.

If the dropsy is extreme, stenosis of the tricuspid orifice is probably also present.

DIAGNOSIS

If aortic regurgitation can be excluded, an apical presystolic murmur presenting the characters previously described is pathognomonic of mitral stenosis.

In those instances, however, in which the signs of aortic incompetence are observed in association with an apical presystolic

thrill and murmur, or murmur only, the diagnosis of mitral narrowing should be made with very great caution.

The two lesions sometimes coexist, but since the auscultatory phenomena of mitral stenosis, so far as the presystolic murmur is concerned, may be exactly simulated (see p. 203) by those of aortic incompetence, it is, under the circumstances mentioned above, often extremely difficult and occasionally impossible to decide positively whether the latter affection is or is not complicated by the former.

The differential diagnosis must be based on a general consideration of the whole of the symptoms and physical signs, assisted by the cardiographic and sphygmographic evidence.

If the presystolic murmur is wanting, as is the case in the final stage of the disease, the short, sharp first sound of mitral stenosis may be mistaken for that of dilatation of the heart. The absence of the second sound at and to the left of the apex would, however establish the diagnosis of mitral stenosis.

A rumbling presystolic murmur is sometimes heard in the mitral area after an attack of pericarditis or in association with pericardial adhesions in the case of children. The murmur heard under these circumstances has not the peculiar vibratory character of the bruit audible in cases of mitral narrowing, nor does it terminate abruptly in the first sound. Furthermore, the first sound does not present the characteristic modification observed in mitral stenosis.

The association of regurgitation through the mitral opening with narrowing of the orifice sometimes leads to difficulty in the detection of the latter affection in the absence of the presystolic murmur.

The diagnosis, in this event, turns largely on the characters of the first sound at the apex, since the effect of regurgitation is to obscure and destroy it, whereas the effect of stenosis is to shorten and exaggerate it. If therefore the first sound at the apex is short and loud, and is followed and not obscured by a systolic bruit, which is neither well conducted into the left axilla, nor audible at the angle of the left scapula, the presence of mitral stenosis may reasonably be suspected. The absence of the second sound at and to the left of the apex would be further evidence in favour of the existence of mitral narrowing.

Moreover, if the symptoms and physical signs of pulmonary congestion and enlargement of the right ventricle, associated with an apical systolic murmur, are more marked than the amount of regurgitation through the mitral orifice, estimated on other grounds, would account for, the possibility of such a complication as mitral stenosis must be taken into consideration.

It appears also that regurgitation through the mitral orifice would occur much more commonly in association with mitral stenosis were it not that the high pressure in the left auricle during the ventricular systole is sufficient to prevent any reflux through the imperfectly closed auriculo-ventricular aperture (Samways).

ESTIMATION OF THE DEGREE OF NARROWING IN MITRAL STENOSIS

The extent of the lesion is determined by a consideration of the following data :—

1. The pulse.—So long as the pulse is regular in force and frequency and the artery is of medium size, the amount of mitral narrowing cannot be very great.

Great reduction in the size of the artery and a small, weak, and extremely irregular pulse are among the indications of severe obstruction.

2. The degree of enlargement of the left auricle.—The increase of dulness in the third and fourth intercostal spaces, immediately to the left of the sternum, will indicate roughly the size of the left auricle.

3. The degree of enlargement of the right ventricle.—The size of this chamber and the force of the ventricular contraction furnish the most reliable evidence of the extent of the mitral narrowing, since it is on this side of the heart and on the left auricle that the stress of the lesion falls.

4. The presence or absence of the aortic second sound at and to the left of the apex.—If the second sound is audible at and to the left of the apex the degree of stenosis is probably slight, for the reasons which have already been given.

The absence of the second sound in this situation is an indication that the narrowing of the orifice is considerable.

5. The amount of accentuation of the pulmonic second sound.—In the absence of pulmonary complications and provided the tricuspid valve is competent, the degree of pronunciation of the pulmonic second sound affords a means of gauging the blood pressure in the lungs, and hence of estimating the degree of obstruction to which the lesion has given rise.

6. The severity of the arterial anæmia and venous engorgement.—So long as there are no complications, and provided the compensatory changes are well developed, the presence of marked arterial anæmia and venous engorgement is significant of serious obstruction.

In order to facilitate the estimation of the extent of the lesion, and to serve as a guide to the prognosis in mitral stenosis, Sir W.

Broadbent has divided the progress of the disease into three stages, which are as follows :—

1. The first stage which is characterised by—
 - (a) A presystolic murmur
 - (b) The gradual development of a short and sharp first sound at the apex
 - (c) Accentuation and reduplication of the pulmonic second sound.
2. The second stage which is characterized by—
 - (a) The disappearance of the second sound at and to the left of the apex
 - (b) A short, sharp first sound
 - (c) A variable diastolic murmur
3. The third stage which is characterized by—
 - (a) The disappearance of the presystolic murmur
 - (b) A short and sharp first sound
 - (c) A tricuspid systolic murmur

In the light of what has already been said with regard to the estimation of the degree of obstruction at the mitral opening, a further explanation of these stages is unnecessary.

It may, however, be pointed out that under this classification, which is based on auscultatory signs, the second and third stages are interchangeable. Thus a case which has reached the third stage may, under favourable conditions and by means of suitable treatment, be restored to the second, a feature that does not obtain with respect to the first two stages.

CHAPTER XIII

AORTIC INCOMPETENCE

Pathogenesis—Morbid Anatomy—Effects on the Heart and Circulation—
Compensation—Sudden Death—Symptoms—Complications—Physical Signs
—Diagnosis—Estimation of the Amount of Regurgitation.

ÆTIOLOGICAL PATHOLOGY

THE chief causes of aortic incompetence are : (1) Acute, sub-acute, or chronic valvulitis of rheumatic, and probably, in some instances, of syphilitic origin ; (2) chronic valvulitis (sclerosis) due to prolonged muscular strain ; and (3) atheroma, which may attack the valve primarily, or as the result of the extension of the morbid process from the root of the aorta.

Syphilis and chronic alcoholism frequently co-operate with mechanical strain in the production of aortic valvular disease ; they are also important factors in the causation of atheroma.

It is often impossible to distinguish between the effects of strain and atheroma, since they are both the result, for the most part, of a common cause, viz. increased arterial tension.

As the result of the operation of one or other of the morbid processes above mentioned, the free margins of the aortic cusps become thickened, rigid, and retracted, or otherwise deformed, so that their proper adaptation is rendered impossible.

The sclerosed tissue is often the seat of ulcerative changes, which may lead to perforation, laceration, or rupture of a flap, and thus to incompetence of the valve. It is also not uncommon to find the thickened segments infiltrated with calcareous salts.

In some instances the proper adaptation of the cusps of the valve is prevented by a growth of vegetations.

Rupture of a segment of a healthy aortic valve is occasionally due to sudden and violent strain. External violence—to wit, a blow on the outside of the chest—has in a few cases been the cause of the rupture of an aortic cusp.

In rare instances aortic incompetence occurs as a congenital lesion.

Dilatation of the root of the aorta may involve the orifice of the vessel, and by this means may give rise to relative incompetence of the valve. The lesion is, however, of rare occurrence.

The valvular changes which lead to aortic incompetence are frequently, indeed usually, accompanied by some induration of the fibrous ring surrounding the orifice of the vessel. The subsequent contraction of the cicatricial tissue leads to more or less constriction of the opening, with the result that valvular incompetence is commonly combined with a variable degree of narrowing of the aortic aperture.

PATHOLOGICAL RESULTS

Effects on the Heart and Circulation

In consequence of the valvular incompetence, a certain quantity of blood flows backwards from the aorta into the left ventricle during its diastole. The ventricle is, therefore, now supplied from two sources, and at the end of diastole its contents exceed the normal by the amount which has returned from the aorta. The accommodation of this additional quantity of blood necessitates an increase in the capacity of the chamber, which can be effected only by the stretching of its elastic walls. This process, if continued, becomes dilatation; hence the earliest effect of aortic incompetence is to produce dilatation of the left ventricle from overfilling. The increased work involved in the propulsion of a larger amount of blood than normal leads to hypertrophy of the ventricular walls, provided, of course, the myocardium enjoys sufficient nutrition.

There is, however, a further and more important cause of dilatation and hypertrophy of the left ventricle in aortic insufficiency, inasmuch as the ventricle has to withstand, during its diastole, the distending effects of the regurgitant stream under the pressure of the arterial recoil.

The strain imposed on the walls of the ventricle by this means will vary with the extent of the lesion and the state of arterial tension. It is probably considerable in all cases, and constitutes the chief source of overwork which has to be performed by the ventricle.

Dilatation would quickly get the upper hand of hypertrophy at the outset were it not that the ventricle falls back on its reserve power, which temporarily staves off the distending effects of the lesion. The subsequent occurrence of hypertrophy restores and maintains the tone of the ventricle, and prevents more dilatation of the chamber than is necessary to accommodate the amount of blood by which it is overcharged.

If hypertrophy did not occur, the reserve power of the heart

would soon become exhausted, and there is then no reason why dilatation of the ventricle should not proceed indefinitely.

The relative proportions of hypertrophy and dilatation of the left ventricle determine in a great measure the adequacy of compensation in aortic incompetence.

The high tension which prevails in the left ventricle during its diastole interferes with the flow of blood through the mitral orifice, and thus leads to a rise of pressure in the left auricle, pulmonic circulation, and right ventricle. The intensity of this process depends largely on the rate of development and extent of the lesion, and on the degree of adequacy of the compensatory changes.

If the valvular incompetence comes on slowly, and is of moderate severity, and provided also that compensation is satisfactory, there is apparently little or no serious interference with the pulmonic circulation. In some instances, however, owing either to the sudden development or to the severity of the lesion, or to the relative or absolute inadequacy of the compensatory changes, or, as most commonly happens, to a combination of these conditions, more or less embarrassment of the pulmonic circulation is produced. The left auricle consequently undergoes dilatation and occasionally some hypertrophy, while the right ventricle usually becomes hypertrophied.

In the late stages of aortic insufficiency, the left ventricle undergoes dilatation from failure, a process that is attended by relative incompetence of the mitral valve whereby the circulation through the lungs is still further hampered.

In many cases, too, the aortic lesion is associated with organic changes in the mitral valve which may render it incompetent.

Dilatation of the right ventricle from failure ultimately supervenes, and the consequent establishment of tricuspid regurgitation is followed by the usual sequence of events, culminating in general venous stasis, visceral congestion, and dropsy.

The most striking and characteristic features of aortic incompetence are, however, observed in connection with its effects on the arterial circulation.

In the first instance the supply of blood to the aorta and peripheral vessels is diminished by the amount which regurgitates into the left ventricle during each diastole, so that a variable degree of arterial anæmia is produced. Provided, however, the dilatation and hypertrophy of the left ventricle are adequate, the quantity of blood thrown into the aorta at each systole exceeds the normal by the amount which flows backwards during diastole, and in this way the ordinary balance of the circulation is restored. The excessive strain to which the arterial system is subjected in consequence of the more forcible injection of a larger quantity of blood than usual into the aorta, has important effects on the walls of these vessels, in that they become stretched and thickened, and frequently show atheromatous changes.

Moreover, the loss of elasticity which these changes entail not only adds still further to the work of the left ventricle, but is also a fertile source of arterial rupture.

The forcible and excessive distension of the peripheral vessels by the powerful systole of the left ventricle, followed by their sudden collapse during diastole, on account of the loss of support to the column of blood at the aortic orifice, gives rise to visible pulsation of the arteries throughout the body. A peculiar vermiform movement of the arterial tubes may also be observed as the pulse wave passes along it.

Under favourable circumstances aortic incompetence may endure for many years without giving rise to any obvious disturbance of the systemic or pulmonic circulation.

Compensation is effected mainly by means of dilatation and hypertrophy of the left ventricle. The dilatation of the chamber affords accommodation for the additional quantity of blood received from the aorta, while the hypertrophy enables the ventricle to cope with the increased work required of it.

When the excess of blood thrown into the aorta at each systole exactly counterbalances the amount of backflow during diastole, compensation is perfect. This result is not uncommonly attained, but in many instances compensation is less satisfactory.

Arterial anæmia and pulmonic congestion develop in proportion to the degree of cardiac insufficiency.

The salutary effects of hypertrophy of the right ventricle and the disastrous results which follow dilatation of this chamber from failure have already been mentioned. The breakdown of compensation in aortic incompetence may be sudden or gradual, and is due to failure of the left ventricle, which undergoes progressive dilatation from incomplete emptying, in consequence of the exhaustion of its muscular walls induced by long-continued overwork.

The series of changes which lead to the final result follow one of two courses. In one event the progressive inadequacy of the left ventricle is accompanied by excessive arterial anæmia and its effects; there is little or no embarrassment of the pulmonic circulation, and dropsy is usually slight or absent. In the other and more common event the increasing dilatation of the left ventricle leads to relative incompetence of the mitral valve followed by pulmonic engorgement, dilatation of the right heart, general venous congestion, and dropsy.

Under either set of conditions the over-distension of the left ventricle may result in sudden arrest of the heart's action in diastole.

Sudden death in aortic incompetence occurs most commonly in those instances in which the lesion arises after middle life, when satisfactory compensation is hardly ever established.

SYMPTOMS

Aortic incompetence, the result of endocarditis, may endure for many years without giving rise to symptoms of any importance.

The lesion, in cases of this kind, is usually inconsiderable, and the compensatory changes in the left ventricle are so perfect that the valvular insufficiency produces no appreciable disturbance of the systemic and pulmonic circulations. It frequently happens, too, under these circumstances, that the patient is able to undergo great physical and mental exertion without any obvious discomfort.

This condition of affairs is, however, the exception rather than the rule, and it is usual, even in well-compensated cases, to find that the subjects of aortic incompetence suffer from undue shortness of breath when any unusual effort is made. At the same time, this symptom is not so pronounced as in mitral affections, owing to the absence of pulmonic congestion in the early stages of aortic disease.

A variable degree of anæmia is noticeable almost from the outset in nearly all cases of aortic insufficiency, and pallor becomes a very marked feature of the terminal stages of the disease.

Epistaxis is sometimes an early and troublesome symptom, and should, in the absence of a definite exciting cause, suggest an examination of the heart.

Throbbing of the carotid arteries and other vessels is often a source of much discomfort to the patient.

Headache, giddiness, faintness, vertigo, sleeplessness, noises in the ears, and flashes of light before the eyes are of common occurrence, and depend upon disturbance of the cerebral circulation.

Præcordial pain may be very severe, and attacks of true angina pectoris are more frequently experienced in aortic incompetence than in any other form of valvular disease.

Evidence of the breakdown of compensation is shown, in the majority of cases, by gradually increasing shortness of breath, palpitation, præcordial pain, and irregularity of the heart's action in association with the signs and symptoms of pulmonic engorgement, portal and systemic venous congestion and dropsy.

The series of events which lead up to the fatal issue do not always take this course, for in a considerable number of instances the signs of "backworking" through the mitral orifice are either absent or but slightly marked, and the patient suffers chiefly from arterial anæmia.

This group of cases is characterized by syncopal and anginoid attacks which are commonly attended by irregularity and intermission of the heart's action. An attack of syncope sometimes terminates in sudden death.

Dyspnœa is easily excited and is apt to come on in paroxysms, but there is no habitual shortness of breath. Sleeplessness is sometimes a prominent and distressing symptom, while vomiting, which is not uncommon, is always a serious feature.

The signs and symptoms of systemic venous engorgement and visceral congestion are not observed, and as a rule dropsy is slight or altogether wanting.

Sudden death, from over-distension and paralysis of the left ventricle, is more commonly observed in aortic incompetence than in any other form of valvular disease. This occurrence is especially to be feared in those cases in which the lesion has arisen during or after middle life, when compensation is seldom or never satisfactory.

Cerebral hæmorrhage consequent on the rupture of a vessel is also a cause of rapid death in aortic incompetence.

The occurrence of aortic regurgitation consequent on the rupture of a valvular cusp is associated with the feeling of something having given way in the chest, and with sudden breathlessness and præcordial anxiety and oppression. The signs and symptoms of rapid dilatation of the heart are subsequently developed in a greater or less degree.

COMPLICATIONS

The complications which may arise in the course of aortic incompetence are to a large extent similar to those described in connection with mitral disease. The points of distinction relate rather to differences in intensity and in the time of occurrence of the various phenomena than to differences in kind.

Thus the signs of pulmonic, portal, and systemic venous congestion are, as a rule, later in their appearance and less marked in their effects in aortic than in mitral lesions, whereas this order of events is reversed with respect to the time of occurrence and degree of severity of systemic arterial anæmia.

Again, angina pectoris is a common complication of aortic incompetence, while, on the other hand, it is very rarely observed in association with mitral disease.

Degeneration of the walls of the blood vessels, which is an important factor in the production of arterial rupture, affects the systemic side of the circulation in aortic insufficiency, and the pulmonic in mitral affections.

The occurrence of embolism is, comparatively speaking, rare in aortic incompetence. Sudden death is more commonly observed in aortic incompetence than in other form of valvular disease. This is accounted for on the grounds that the forces which lead to over-distension of the left ventricle operate more powerfully in aortic insufficiency than in any other valvular affection.

The only other complication of aortic incompetence to which special reference need be made is the occasional occurrence of collapse of the lower lobe of the left lung in consequence of the pressure exerted upon it by the enlarged left ventricle.

PHYSICAL SIGNS

Physiognomy.—The face is usually very pale or greyish white in colour and often wears a somewhat anxious expression.

The skin and mucous membranes are anæmic.

Pulsation may be observed in the arteries of the neck, head, and other parts, and is often accompanied by a visible locomotion of the vessels.

The development of mitral regurgitation in the course of aortic incompetence may give rise to some cyanosis of the lips and face, but pallor usually predominates even under these circumstances.

Pulse.—The pulse is slightly increased in frequency, but remains regular in force and rhythm until the heart fails.

The artery is large, but empty between the beats, and the vessel wall is usually more or less thickened and tortuous.

The pulse wave is sudden, large, and sometimes vibratory; its duration is very short; it rises quickly and falls remarkably abruptly, so that it acquires the collapsing character so peculiar to the pulse of aortic regurgitation. Hence the terms "collapsing pulse," "water-hammer pulse," etc., which have been used to describe it.

So long as the heart is acting powerfully the peculiar features of the radial pulse are exaggerated by raising the arm.

The collapsing character of the pulse is often most readily appreciated by grasping the fleshy part of the raised forearm of the patient with one or both hands.

Another feature of considerable interest with regard to the pulse in aortic incompetence is the loss of time manifested between the systole of the left ventricle and the appearance of the pulse wave at the wrist. This phenomenon, which is not confined to the pulse of aortic regurgitation, is probably due, according to Professor Allbutt (quoting Chapman), to prolongation of the systole of the left ventricle.

The collapsing character of the pulse is explained by the rapid and forcible injection of a much larger quantity of blood than usual into the aorta and systemic arteries, and the sudden emptying of these vessels in consequence of the backward flow into the left ventricle and the onward flow into the capillaries.

The dilatation of the arteries, like that of the ventricle, is a compensatory change, since it provides room for the increased quantity of blood which the systemic vessels must accommodate in order to neutralize the effects of the regurgitation through the aortic orifice.

The dilatation of the small arteries in conjunction with the forcible action of the hypertrophied left ventricle is the source of the phenomenon known as capillary pulsation, which is observed in the skin, nails, and mucous membranes in some cases of aortic incompetence. It is due to the failure of the dilated arteries to obliterate the pulse wave, which in consequence penetrates as far as the capillaries, and in some instances as far as the peripheral veins, with the production of venous pulsation.

Capillary pulsation is most readily elicited by gentle friction of

the skin until it becomes reddened, when it will be noticed that the colour fades with each diastole and reappears with each systole of the heart.

The *pulsus bisferiens* is sometimes found in association with aortic incompetence.

Pressure with the stethoscope over a large artery, such as the carotid, brachial, or femoral, etc., sometimes gives rise to a to-and-fro murmur at the site of constriction, consequent on the formation of a fluid vein, which is produced not only by the onward flow during systole, but also by the backward flow towards the left ventricle during diastole.

The systolic and diastolic elements of the bruit may occur separately, and the presence of the latter is always significant of extensive regurgitation through the aortic orifice.

HEART

Inspection.—The cardiac impulse is diffused, and there is not uncommonly some bulging of the præcordial region. The apex beat is displaced downwards and outwards. Systolic recession of the intercostal spaces in the neighbourhood of the apex beat is sometimes observed.

Palpation.—The impulse of the left ventricle is unusually powerful, and conveys the sensation of a forcible heave or thrust. A diastolic impulse is sometimes felt at the apex when the amount of regurgitation is excessive, and the left ventricle has undergone considerable dilatation.

A diastolic thrill is occasionally observed over the base of the heart, and in rare instances a presystolic thrill can be felt in the region of the apex.

Percussion.—The area of cardiac dulness is increased in all directions, but chiefly downwards and to the left, in correspondence with the hypertrophy and dilatation of the left ventricle.

The right limit of cardiac dulness rarely extends much beyond the right sternal edge, and commonly lies within this line.

Auscultation.—At the base of the heart, over the aortic cartilage, the second sound is found to be wholly or partially replaced by a diastolic murmur, which is propagated directly downwards along the sternum as far as the ensiform cartilage, or obliquely downwards towards the apex. In the upward direction it may be audible as high as the sterno-clavicular articulation, or even higher. The murmur is usually soft and blowing in character, but in some instances it acquires a musical quality, while in others it is loud, rough, and vibratory. It may occupy the whole or a portion only of the diastolic period, and as a rule it tails off towards its termination.

The site of maximum intensity of the murmur varies in different

cases. In some it is heard best about the mid sternum, or along the left sternal edge between the third and fifth ribs; in others it is loudest over the ensiform cartilage or at the apex.

The variability in the site over which the murmur is most distinctly audible depends mainly on differences in the position (*i.e.* the particular cusp or cusps affected), character, and extent of the valvular lesion, whereby the direction of the regurgitant stream and the vibratile qualities of the tendinous structures composing the valve are materially altered.

The condition of the aortic second sound requires careful investigation. The presence or absence of the sound furnishes important evidence in the estimation of the amount of regurgitation.

In order to avoid the possibility of confusing the aortic with the pulmonic second sound the stethoscope should be placed over the carotids in the neck, where the latter sound is inaudible.

So long as the aortic sound is heard, or, in other words, so long as the segments of the valve are capable of giving rise to audible vibrations, they must still act as a check to the backflow of blood into the left ventricle, and therefore the amount of regurgitation cannot be very great.

On the other hand, absence of the aortic second sound is among the signs of serious leakage.

The first sound at the base of the heart over the aortic cartilage may be unaltered, or it may be more or less replaced by a systolic murmur, due to coexisting stenosis of the aortic opening or to dilatation of the root of the aorta.

A systolic bruit in this situation may depend also on anæmia, on roughening of the aortic cusps, or on aortitis.

The differential diagnosis of the systolic element of a combined systolic and diastolic murmur, audible over the aortic cartilage, will be considered under aortic stenosis.

At the apex the first sound is usually somewhat dull and muffled, and it may be accompanied by a systolic bruit, which is due either to the muscular and relative incompetence of the mitral valve, attending dilatation of the left ventricle, or to concurrent structural disease at this orifice.

The diastolic murmur is not uncommonly audible at the apex, and in some instances it so closely simulates the presystolic bruit, observed in cases of mitral stenosis, that the exclusion of the latter lesion is almost impossible.

The cause of the phenomenon in aortic incompetence is still a matter of doubt, but it has been explained by supposing that the large anterior flap of the mitral valve is thrown into vibration either by the two blood currents which flow against its opposite sides, or by the direct impact of the regurgitant stream from the aorta. Another view (that of Flint) suggests that the rapid filling of the left ventricle from two sources floats up the mitral curtains, so that at the time of the auricular systole there is a virtual stenosis of the

mitral orifice, and the current of blood flowing between the opposed segments of the valve, throws them into vibrations, whereby the characteristic murmur is produced.

In whatever manner produced, the presence of a presystolic thrill and bruit at the apex, in association with well-marked signs of aortic incompetence, is by no means conclusive of the coexistence of mitral narrowing. In cases of this kind the diagnosis of mitral stenosis must be based on a careful consideration of the concomitant signs and symptoms of the condition.

DIAGNOSIS

The diagnosis of aortic incompetence seldom gives rise to much difficulty, and depends chiefly on the association of an aortic diastolic murmur with enlargement of the left ventricle and a collapsing pulse.

In the absence of the diastolic murmur, an event of very rare occurrence, the peculiar characters of the pulse and the alteration in size of the left ventricle would suffice for the recognition of the lesion.

The site of maximum intensity and the area of distribution of the bruit, taken in conjunction with the concomitant changes in the heart and pulse, serve to distinguish it from diastolic murmurs produced in other situations.

The differential diagnosis of the nature of the lesion lies for the most part between endocarditis and atheromatous disease, since, in the large majority of cases, aortic incompetence, whether it occurs alone or in combination with stenosis, is due to the one or to the other of these morbid processes. In attempting to distinguish between them, the age, sex, occupation, and history of the patient, as well as the associated symptoms and physical signs, must be taken into consideration.

In young people and women, and especially in those cases in which there is a definite history of acute or chronic rheumatism, the probabilities are greatly in favour of endocarditis.

On the other hand, if the subject is a man of middle age, in whom there is evidence of overwork, strain, gout, syphilis, alcoholism, chronic renal disease, etc., and no history of rheumatism, the lesion may reasonably be ascribed to atheroma.

The differentiation between the two forms of disease producing aortic incompetence is, however, more accurately and certainly made by means of the physical signs.

Thus the pulse of aortic regurgitation due to atheromatous disease of the aortic valve shows little or no real collapse of the artery between the beats. The vessel is large, firm, and tortuous, and its walls are more or less thickened. The pulse wave comes and goes

quickly, and though easily compressible, the artery can commonly be felt between the beats.

The absence of collapse in the pulse is accounted for by the comparatively small amount of regurgitation which of necessity must obtain in atheromatous affections of the aortic valve, since any large amount of valvular insufficiency from this cause would be incompatible with life.

Furthermore, the retardation of the pulse wave between the heart and peripheral arteries is much less marked in aortic incompetence due to atheroma than in that caused by endocarditis.

As regards the cardiac physical signs, it may be noted that while a combined murmur (*i.e.* systolic and diastolic) may occur in both forms of the disease, it is more commonly found in association with atheromatous than with inflammatory affections of the aortic valve.

Of much greater importance, however, is the character of the aortic second sound. As already pointed out, this sound is impaired or lost in that form of valvular insufficiency which is produced by endocarditis. In atheromatous lesions at the aortic opening accompanied by incompetence of the valve, the second sound is often loud and ringing in quality, and is heard immediately in front of the diastolic bruit.

The presence of the symptoms or signs of dilatation or aneurism of the aorta, in addition to those of aortic incompetence, would afford strong evidence in favour of an atheromatous lesion. Attacks of cardiac pain or angina pectoris disproportionate to the severity of the lesion, estimated on other grounds, would point in the same direction.

It must, however, be borne in mind that the two forms of disease may coexist, for, in many instances, a lesion originally due to endocarditis becomes the seat of atheromatous changes and *vice versa*. In this way a differential diagnosis may be rendered impossible.

THE ESTIMATION OF THE AMOUNT OF BACKFLOW IN AORTIC INCOMPETENCE

The following are the chief sources of information :—

1. **The character of the pulse.**—The suddenness, rapidity, and extent of the arterial collapse, the size of the vessel, and its condition between the beats are the points to which attention should be chiefly directed.

If the artery is emptied gradually, and especially if it can be felt between the beats, and is of moderate size, the amount of regurgitation is inconsiderable, even though the pulse wave be sudden and forcible.

The more rapid and complete the collapse of the pulse the greater is the amount of regurgitation.

Among the other indications of severe incompetence are well-

marked capillary or venous pulsation, and the presence of a diastolic murmur in the peripheral arteries.

A visible pulse in the superficial peripheral arteries such as the carotids, radials, etc., invariably accompanies serious insufficiency of the aortic valve, provided the left ventricle is not failing.

2. The extent and character of the enlargement of the left ventricle.—In the absence of the symptoms or signs of circulatory embarrassment the degree of hypertrophy and dilatation of the left ventricle is, to a large extent, a measure of the severity of the valvular incompetence.

If the heart is failing, hypertrophy has more significance than dilatation, but it cannot be said that either condition has much weight in the estimation of the amount of regurgitation under these circumstances.

3. The presence or absence of the second sound in the neck.—If the second sound is audible over the carotid arteries in the neck, it follows, for the reasons previously given, that the amount of back-flow into the left ventricle cannot be large. On the other hand, the absence of the second sound in this situation is one of the signs of serious incompetence.

4. The character of the murmur.—The information afforded by the character of the murmur is of very little value.

As a rule, a loud murmur signifies that the heart is acting with vigour, and therefore the probabilities are that the amount of leakage is not very great.

Severe incompetence may be associated with a short, soft, diastolic bruit.

CHAPTER XIV

AORTIC STENOSIS

Pathogenesis—Morbid Anatomy—Effects on the Heart and Circulation—Compensation : its duration and failure—Symptoms—Complications—Mode of Termination—Physical Signs—Diagnosis—Estimation of degree of Stenosis.

ÆTIOLOGICAL PATHOLOGY

AORTIC stenosis arises under precisely the same pathological conditions as aortic incompetence. The chief causes, therefore, of aortic stenosis are : (1) endocarditis due to rheumatism, the zymotic fevers, syphilis, etc. ; (2) endocarditis due to prolonged muscular strain ; (3) atheroma which may attack the aortic valve primarily, or, as most commonly happens, by extension from the aorta.

Aortic stenosis is occasionally observed as a congenital malformation.

The morbid changes lead to thickening, rigidity, and retraction of the semilunar cusps, with more or less induration and constriction of the fibrous aortic ring.

The segments of the valve sometimes become adherent to one another, and thus diminish the size of the orifice, or they may be the seat of abundant vegetations, which obstruct the passage of blood through the opening.

Atheromatous disease of the valve sometimes leads to more or less complete calcification of the semilunar cusps and basal ring. The cusps of the valve are consequently unable to fall back completely during the ventricular systole, so that a variable degree of obstruction is produced at the aortic opening.

In a very large proportion of cases aortic stenosis is combined with incompetence of the valve, owing to the inability of the thickened and rigid segments to adequately close the narrowed orifice.

Indeed, pure aortic obstruction is one of the rarest of cardiac lesions, but it will be convenient to include in this description those forms of aortic disease in which stenosis is the predominant feature.

PATHOLOGICAL RESULTS

Effects on the Heart and Circulation

The effect of aortic stenosis is to interfere with the passage of blood from the left ventricle into the aorta during systole.

The difficulty experienced by the left ventricle in the expulsion of its contents calls for an increased display of force, which, under favourable circumstances, leads to hypertrophy of its walls. If, by this means, the volume of blood propelled into the aorta during each systole does not fall below the normal, compensation is established. It will therefore appear that in aortic stenosis compensation depends solely on hypertrophy of the left ventricle.

If the constriction of the opening is attended by incompetence of the valve, a variable degree of dilatation of the left ventricle is also produced, for the reasons which were given in the previous chapter.

Uncomplicated aortic stenosis may endure for many years without giving rise to any appreciable disturbance of the circulation; indeed, it sometimes happens that the lesion is not discovered until after death from other causes.

Compensation is maintained longer in aortic stenosis than in any other form of valvular disease, but in the absence of a definite exciting cause failure of the heart is rarely recovered from.

The breakdown of compensation usually depends on progressive constriction of the aortic orifice, or on interference with the blood supply to the heart through the coronary arteries, or on the occurrence of a complication.

The left ventricle becomes unable to completely empty itself at each systole, and consequently undergoes dilatation from incomplete emptying, *i.e.* from failure.

A variable degree of arterial anæmia ensues, and the dilatation of the left ventricle, if continued, leads to muscular and relative incompetence of the mitral valve, followed by pulmonic engorgement, dilatation of the right heart, general venous congestion, and dropsy.

The occurrence of mitral regurgitation in the course of aortic stenosis may depend also on chronic forcing of the mitral valve by the high pressure which prevails in the left ventricle, or on concomitant structural disease at the mitral orifice.

SYMPTOMS

So long as compensation is good, the subjects of aortic stenosis remain free from symptoms until the degree of narrowing becomes sufficiently great to seriously interfere with the supply of blood to the aorta and systemic vessels. The patient then begins to suffer from headache, giddiness, syncopal attacks, and other symptoms of disordered cerebral circulation.

Anæmia is developed, and the defective supply of blood to the systemic arteries leads to interference with nutrition and coldness of the extremities associated with general nervous and muscular debility.

As compensation begins to fail, dyspnœa, palpitation, pain, which may have anginal characters, and other symptoms of cardiac insufficiency make their appearance.

The dilatation of the left ventricle which accompanies failure of compensation is attended by regurgitation through the mitral orifice, together with the signs and symptoms of pulmonic and systemic venous congestion.

As a rule death takes place slowly from gradual failure of the ventricles. A sudden termination is sometimes observed as the result of syncope, embolism, or the rupture of a cerebral vessel.

The complications of aortic stenosis do not require special consideration, since they resemble, for the most part, those described in connection with aortic incompetence.

PHYSICAL SIGNS

Physiognomy.—The appearance presented by the subjects of aortic stenosis is in no way characteristic until the disease has made considerable progress, when the signs of anæmia are gradually developed. The complexion then acquires the sallow or greyish-white hue described under aortic regurgitation, and the skin and mucous membranes become pallid.

A bluish tinge affecting the lips, cheeks, and extremities, etc., may be noticed after failure of the left ventricle has occurred, and is due to insufficient aëration of the blood following leakage through the mitral valve and the consequent production of pulmonic congestion.

Pulse.—So long as compensation is maintained the pulse is slow and regular both in force and frequency. The artery is small and can be felt between the beats, but, as a rule, is easily compressible.

The pulse wave is small, long, and well sustained; it rises gradually and falls slowly.

Both the anacrotic and the bisferiens pulse may be found in association with aortic stenosis, but it cannot be said that either pulse is pathognomonic of this lesion.

The exact significance of these pulses has not yet been fully worked out, but, speaking generally, the indication in either event is organic disease at the orifice or along the course of the aorta.

HEART

Inspection.—Bulging of the præcordial region is not uncommon. The apex beat is displaced downwards and slightly outwards.

Palpation.—The impulse of the left ventricle conveys to the hand the impression of a slow, deliberate, forcible thrust. A thrill, systolic in time, is sometimes palpable over the base of the heart.

Percussion.—The area of cardiac dulness is increased chiefly downwards and to the left, and corresponds with the enlargement of the left ventricle.

Auscultation.—At the base of the heart, over the aortic cartilage, the first sound is accompanied, or more or less replaced, by a loud, rough, and rasping murmur, which is conducted upwards along the right sternal edge into the neck. The murmur is usually audible over the upper portion of the thorax and along the course of both carotid arteries. It can frequently be heard at the apex, and occasionally over the whole præcordium.

The murmur usually has the characters above indicated, but in some instances it is soft and blowing, in others musical.

As a rule the bruit occupies the whole of the interval between the commencement of the first sound and the occurrence of the second sound. The second sound over the aortic cartilage is commonly muffled and indistinct, and is sometimes more or less replaced by a diastolic murmur.

The first sound at the apex is usually dull and ill defined, and may be accompanied by a systolic bruit of mitral origin.

The establishment of mitral regurgitation, which, as already explained, takes place sooner or later in the course of aortic stenosis, is followed by the signs and symptoms of pulmonic engorgement, portal and systemic venous congestion, and dropsy.

The occurrence of this train of events in association with aortic stenosis, in the absence of a definite existing cause, indicates failure of the left ventricle and the breakdown of compensation, and is usually of the worst possible augury.

DIAGNOSIS

In the diagnosis of aortic stenosis much more importance attaches to the character of the pulse and to the changes in the left ventricle than to the presence of a systolic murmur in the aortic area, inasmuch as the latter sign may be observed in various other conditions, chief among which are rigidity, roughening, or fenestration of one or more of the cusps of the aortic valve, dilatation or aneurism of the aorta, anæmia, and acute or sub-acute aortitis.

A systolic basic murmur, due to the roughening and rigidity of the aortic cusps, that is caused by atheroma is almost certainly accompanied by accentuation of the second sound, and is therefore not likely to be mistaken for the bruit of aortic stenosis. Again, fenestration of an aortic cusp, or a shred of fibrin hanging from some portion of the valve, would not give rise to any modification of the pulse, or to enlargement of the left ventricle.

In dilatation of the aorta the volume of the pulse is larger than in aortic stenosis. Moreover, the presence of pulsation and dulness in the intercostal spaces to the right of the sternum above the level of the third or second rib, together with the well-marked and charac-

teristic accentuation of the second sound, which is invariably observed in aortic dilatation so long as the aortic valve is competent, would suffice for the recognition of the lesion.

The presence of pressure signs would establish the diagnosis of aortic aneurism.

The systolic aortic murmur due to anæmia is soft and blowing in character, and does not substitute itself for the first sound, as in aortic stenosis. Moreover, the murmur in the aortic area is usually, if not always, preceded and accompanied by a venous hum in the neck and a systolic bruit in the pulmonic area. Furthermore, the age and general condition of the patient, together with the absence of the signs of cardiac hypertrophy, or of any special modification in the volume of the pulse, renders the differential diagnosis between anæmia and aortic obstruction comparatively easy.

The determination of the nature of the lesion producing aortic stenosis depends on the age, sex, and history of the patient, and on the condition of the peripheral vessels. The cardiac physical signs do not, as a rule, afford much information in this respect, though Constantin Paul considers that the systolic bruit of aortic obstruction due to atheroma tends to spread laterally rather than vertically.

If the obstruction at the aortic opening caused by atheroma is due merely to rigidity of the segments of the valve, the second sound is usually accentuated.

The appearance of an aortic systolic murmur accompanied by irregular pyrexia, retrosternal pain and rapid cardiac failure would be strongly suggestive of aortitis.

ESTIMATION OF THE DEGREE OF NARROWING IN AORTIC STENOSIS

The degree of constriction in aortic stenosis is determined by means of the character of the pulse, and the extent of the hypertrophy of the left ventricle.

1. **The pulse.**—A moderate degree of arterial constriction with a pulse of fair size is compatible with an inconsiderable amount of narrowing at the aortic orifice. This inference is strengthened if it is found that a sudden additional strain thrown on the heart leads merely to increased force and frequency of the pulse.

On the other hand, a small vessel with great diminution in the volume of the pulse is indicative of severe aortic obstruction.

2. **The degree of hypertrophy of the left ventricle.**—So long as compensation is maintained, the extent of the ventricular hypertrophy is a measure of the degree of stenosis which obtains. Nevertheless it cannot be too strongly insisted upon that the indications afforded by the pulse altogether outweigh the evidence supplied by the condition of the left ventricle in estimating the degree of narrowing in cases of aortic stenosis.

CHAPTER XV

TRICUSPID INCOMPETENCE

Pathogenesis—Morbid Anatomy—Effects on the Heart and Circulation—Symptoms—Physical Signs—Diagnosis—Estimation of the amount of Regurgitation.

ÆTIOLOGICAL PATHOLOGY

Regurgitation through the tricuspid opening may be due to—

1. Organic disease of the segments of the valve
2. Muscular or relative incompetence of the valve

Tricuspid insufficiency due to structural disease of the valve is of comparatively rare occurrence. It may be produced by acute, sub-acute, or chronic endocarditis, arising either “in utero” or during after life. In some instances the insufficiency of the valve is due to degenerative processes.

The valvular incompetence is brought about by changes similar to those described in connection with mitral regurgitation, and in the large majority of cases the affection of the tricuspid valve is secondary to endocarditis on the left side of the heart.

Byrom Bramwell is of the opinion that acute inflammation of the tricuspid valve is much more common than is generally supposed, and, furthermore, that the inflammatory process usually subsides without giving rise to any permanent structural changes.

Relative incompetence of the tricuspid valve is primarily dependent on dilatation of the right ventricle, the result of the myocardial debility and degeneration associated with cardiac overstrain, anæmia, the acute specific fevers, and malnutrition from other causes.

It is observed, therefore, in connection with affections of the pulmonic valve. It is also a common sequel to lesions of the mitral valve, and sooner or later it arises in all forms of lung disease which lead to obstruction in the pulmonic circulation.

The mechanism of production of this form of valvular incompetence has already been fully considered in connection with mitral regurgitation.

PATHOLOGICAL RESULTS

Effects on the Heart and Circulation

In consequence of the incomplete closure of the tricuspid valve, blood is forced backwards into the right auricle during the ventricular systole. The auricle, now supplied from two sources, undergoes dilatation from overfilling and its walls subsequently hypertrophy, owing to the additional work entailed in the propulsion of a larger quantity of blood than usual. The latter process is seldom very pronounced, partly because the auricular walls are incapable of much hypertrophy, and partly because the conditions necessary for the development of hypertrophy are rarely satisfactory in cases of tricuspid incompetence.

As the result of the increase in the capacity of the right auricle, a larger quantity of blood than normal is propelled into the right ventricle during diastole. Dilatation of the right ventricle from overfilling is thereby produced, and this process, in an *uncomplicated* case of tricuspid regurgitation, is followed by hypertrophy of the chamber from increased work. Since in the large majority of instances, however, the limits of hypertrophy have already been reached, the former effect alone is usually observed.

The high pressure which prevails in the right auricle, especially during the ventricular systole, offers a serious obstacle to the return of blood to the heart, so that congestion and ultimately engorgement of the portal and systemic venous circulations is produced. Moreover, the great venous trunks are exposed to the direct effects of the regurgitant stream from the ventricle, which not only intensifies the congestion, but leads also to distension and dilatation of these vessels.

It will be unnecessary to discuss here the effects of portal and systemic engorgement on the abdominal and other viscera and peripheral tissues, inasmuch as they were fully considered in connection with mitral incompetence.

It may, however, be again pointed out that hepatic enlargement is an early and characteristic feature of tricuspid regurgitation.

Incompetence of the tricuspid valve is also accompanied by a greater or less degree of arterial anæmia consequent on the diminished supply of blood to the lungs, and hence to the left heart and aorta.

SYMPTOMS

In uncomplicated cases regurgitation through the tricuspid orifice may be unattended by symptoms of any moment, provided the amount of leakage is very slight. Any considerable degree of tricuspid insufficiency gives rise to symptoms which are similar.

for the most part, to those described in connection with the final stages of mitral disease.

The onset of tricuspid regurgitation during the course of left-sided cardiac lesions is evidenced by an increase of dyspnoea, by cyanosis and dropsy, and by the symptoms significant of visceral engorgement.

The shortness of breath is usually a prominent symptom, and is aggravated by the occurrence of pulmonic complications, of which hydrothorax and bronchial catarrh are the most important.

Dropsy commences with œdema of the feet and ankles and gradually spreads upwards. In severe cases the serous cavities, as well as all the subcutaneous tissues, are involved, so that the patient becomes more or less completely waterlogged.

The congestion of the portal circulation is shown by a feeling of weight, heaviness, or tenderness in the right hypochondrium, by digestive disturbances, and by irregular action of the bowels. Hæmatemesis and melæna occasionally occur, and are attributable to the high pressure in the portal vessels.

Headache, giddiness, vertigo, and insomnia are among the indications of the disturbance of the circulation through the brain.

The urine is scanty, high coloured, and usually throws down, on cooling, a copious deposit of lithates. It usually contains also a small quantity of albumen, and occasionally a little blood.

Venous thrombosis may lead to embolism of the pulmonic artery, and in this way to sudden death. The usual mode of termination of these cases is by gradual failure of the right ventricle.

In those instances in which tricuspid incompetence is due to a temporary or curable cause, such as cardiac overstrain, anæmia, the acute specific fevers, etc., the symptoms do not necessarily present the serious characters described above.

PHYSICAL SIGNS

Physiognomy.—The subjects of tricuspid incompetence present appearances similar to those observed in the terminal stages of mitral disease. Thus the face is dusky or livid, the lips become cyanosed, and the limbs and trunk are more or less œdematous.

In other instances the signs of anæmia are obtrusive, but in any event dropsy is usually an early and prominent feature.

Pulse.—Tricuspid incompetence, *per se*, produces little alteration in the character of the pulse. The modifications which may be observed are due, for the most part, to the lesions on the left side of the heart, whereon the tricuspid affection depends.

With this reservation, it may be stated that the pulse in tricuspid incompetence is diminished in volume and is usually irregular both in force and frequency.

The artery is small and cannot be felt between the beats, while the pulse wave is short, weak, and badly sustained.

A reduction in the size of the right radial pulse as compared with the left has been observed, and is explained on the assumption that pressure is exerted on the right subclavian artery by the distended right auricle and enlarged veins.

HEART

Inspection.—Examination of the neck shows more or less distension of the jugular veins, which will frequently fill from the cardiac side when emptied by pressure with the finger from below upwards.

Pulsation is also usually observed in the jugular veins when the amount of regurgitation is considerable. The pulsation is commonly systolic, and is due to the reflux wave along the jugulars that is produced by the ventricular contraction. In some instances the auricular contraction also gives rise to a reflux current along the veins, so that a double jugular pulsation (*i.e.* presystolic and systolic) is observed. It must be borne in mind, however, that jugular pulsation is not pathognomonic of tricuspid incompetence, nor is the presence of the lesion excluded by the absence of the sign.

The regurgitant stream through the tricuspid orifice is likewise transmitted into the liver, by way of the right auricle, inferior vena cava, and hepatic veins, with the result that systolic expansile pulsation of this organ may also be produced.

Epigastric pulsation is usually well marked, and is due to the movements of the enlarged right ventricle.

Pulsation is less commonly seen in the third and fourth right intercostal spaces close to the sternum. This sometimes depends on the contraction of the right auricle, but in the majority of cases it is produced by the reflux current from the ventricle into the auricle.

Palpation.—The epigastric impulse is forcible and heaving until the right ventricle fails.

Pulsation may be detected over the liver. Care must be taken, however, not to mistake the impulse communicated to the liver by the systole of the right ventricle for true hepatic pulsation.

Percussion.—The area of cardiac dulness is increased, especially towards the right, in consequence of the enlargement of the right auricle and ventricle.

Extension of dulness to the left is also commonly observed as the result of concurrent disease on the left side of the heart.

The lower limit of hepatic dulness may extend down to, or even below, the level of the umbilicus.

Auscultation.—Tricuspid incompetence is commonly attended by a soft and blowing systolic murmur, which partially or wholly obscures the first sound. The site of maximum intensity of the murmur is usually about the junction of the fifth and sixth left costal cartilages with the sternum, or over the ensiform cartilage.

The bruit is propagated upwards and to the right, and outwards it may be heard as far as the apex.

The absence of the murmur does not exclude the existence of tricuspid incompetence.

The pulmonary second sound is weak, or diminished in intensity if previously accentuated.

Reduplication of the second sound at the base is also commonly observed.

DIAGNOSIS

A systolic murmur audible in the tricuspid area may be the sole evidence of slight leakage through the right auriculo-ventricular opening. The precise significance of such a murmur would be determined by attention to its site of maximum intensity, and direction of transmission.

If the signs of enlargement of the right heart and of the liver are observed in association with a systolic murmur in the tricuspid area, the presence of tricuspid incompetence is hardly open to doubt. The diagnosis would be confirmed by venous pulsation in the neck, or by pulsation of the liver. Moreover, the diagnostic significance of these two signs is not weakened by the absence of the systolic murmur. In endeavouring to ascertain whether tricuspid incompetence is primary, or secondary, it is necessary to take into consideration the circumstances under which the lesion has arisen, as well as the condition of the left heart and pulmonic circulation.

THE ESTIMATION OF THE AMOUNT OF REGURGITATION IN TRICUSPID INCOMPETENCE

In attempting to estimate the amount of regurgitation in tricuspid incompetence the observer must be guided by the general principles laid down in the foregoing chapters.

The precise estimation of the amount of leakage is of less importance than the determination of the exact causes which have led to the occurrence of the lesion.

Thus tricuspid regurgitation, secondary to disease of the lungs or left heart, is always an event of serious significance, yet the gravity of the outlook is but slightly affected by the degree of insufficiency which obtains.

Briefly, the chief points to be considered in the estimation of the amount of regurgitation in tricuspid incompetence are :—

1. The degree of enlargement of the right auricle and ventricle.
2. The presence or absence of venous pulsation in the neck.
3. The presence or absence of pulsation of the liver and the degree of enlargement of the organ.
4. The degree of arterial anæmia.

CHAPTER XVI

TRICUSPID STENOSIS

Pathogenesis—Morbid Anatomy—Effects on the Heart and Circulation—Symptoms—Physical Signs—Diagnosis—Estimation of degree of Stenosis.

ÆTIOLOGICAL PATHOLOGY

TRICUSPID stenosis occurs in rare instances as a congenital malformation, when it is almost invariably associated with other developmental anomalies of the heart, as, for instance, patency of the foramen ovale, incomplete ventricular septum, stenosis of the pulmonary artery, etc.

The acquired form of the disease is more common in women than men, and is usually secondary to and probably dependent on stenosis at the mitral orifice for the following reasons:—

1. The almost constant association of the two lesions in adults.
2. Both lesions occur more commonly in women than men, the ratio in each case being about the same, *i.e.* as three or four to one. No such disproportion exists between the sexes in the liability of the heart to right-sided endocarditis apart from this association of mitral and tricuspid stenosis. The inference is, therefore, that the relationship between the two lesions is causal.
3. The remarkable fact that the degree of narrowing at the mitral opening is always greater than that at the tricuspid orifice.

If it be admitted that the two lesions are related in the manner suggested, the occurrence of tricuspid stenosis is explained by the long-continued strain and irritation to which the valve is exposed in consequence of the rise of pressure in the right ventricle following the obstruction at the mitral orifice. This irritation might lead to chronic thickening, whether inflammatory or otherwise, of the structures forming the tricuspid orifice and valve, which under suitable conditions would terminate in stenosis of the tricuspid orifice.

The degree of narrowing at the tricuspid opening is less than

that at the mitral orifice, because of its later onset and consequently shorter duration.

Tricuspid stenosis is sometimes, though rarely, observed as a solitary lesion, and in such cases it is reasonable to suppose that the affection is the sequel of a right-sided endocarditis, which, as Byrom Bramwell has urged, is probably more common than is generally supposed. The morbid changes which give rise to narrowing of the tricuspid orifice are similar, for the most part, to those observed in obstruction at the mitral opening.

PATHOLOGICAL RESULTS

Effects on the heart and circulation.—The interference with the flow of blood through the tricuspid orifice increases the work of the right auricle, and thereby gives rise to hypertrophy of its walls. A certain amount of dilatation of the chamber also takes place, probably because the degree of hypertrophy is insufficient to enable the auricle to completely empty itself.

Moreover, stenosis of the tricuspid opening is usually accompanied by more or less incompetence of the valve, so that dilatation of the auricle may, in part at least, be due to overfilling.

The enlargement of the right auricle in tricuspid stenosis is commonly very remarkable.

Engorgement of the portal and systemic venous circulations is an early and prominent feature of the disease, and is produced in the manner described in previous chapters.

The diminished supply of blood to the lungs, left heart, and aorta leads to a greater or less degree of arterial anæmia. The hypertrophy and dilatation of the right ventricle which is usually observed is due to the concurrent narrowing of the mitral opening. The presence of pulmonic congestion depends upon the same cause.

SYMPTOMS

The symptoms resemble those of mitral disease, complicated by tricuspid incompetence. Infra-mammary or epigastric pain is frequently a very pronounced symptom, and it may be associated with palpitation and great dyspnoea.

PHYSICAL SIGNS

Physiognomy.—Cyanosis of the face, lips, and ears is commonly observed, and the extremities are usually cold, livid, and more or less oedematous.

Dropsy is present in a variable degree in every case, and it may amount to complete waterlogging of the patient. In this respect tricuspid stenosis offers a marked contrast to mitral obstruction,

which *per se* seldom gives rise to much dropsy. Indeed, so rarely is this the case that, as Sir William Broadbent has pointed out, the occurrence of severe dropsy in the course of uncomplicated mitral stenosis justifies a diagnosis of concurrent obstruction at the tricuspid opening.

Pulse.—The pulse of tricuspid obstruction presents the characters described under mitral stenosis.

Heart.—The jugular veins are distended and turgid, and the liver is enlarged, but pulsation is not, as a rule, observed in either situation. If, as sometimes happens, jugular pulsation can be detected, it is due to the reflux current that is produced by the auricular systole.

Epigastric pulsation is usually well marked, and the impulse of the right ventricle is forcible until failure of the heart supervenes.

A presystolic thrill can sometimes be detected in the tricuspid area.

The area of cardiac dulness is increased laterally, and may extend from an inch to an inch and a half outside the right sternal edge.

The dulness is most marked in the third, fourth, and fifth right intercostal spaces.

A presystolic or diastolic bruit may be heard in the tricuspid area, and it is usually accompanied by a soft blowing systolic murmur, which *follows* and does not obscure the first sound. The presystolic bruit is exceedingly inconstant, and may be easily overlooked unless repeated and careful auscultation of the tricuspid area be carried out.

The sounds at the base of the heart are usually weak.

DIAGNOSIS

If the signs of enlargement of the right heart are observed in conjunction with a presystolic or diastolic bruit, which is limited to the tricuspid area, a diagnosis of tricuspid stenosis can be made without hesitation.

As a rule, however, it is extremely difficult, and often impossible, to decide that the murmur heard in the tricuspid area is not due to the conduction of the concurrent presystolic bruit from the mitral region. It is therefore necessary to search for other evidence of tricuspid obstruction.

If mitral regurgitation can be excluded, the occurrence of serious dropsy in the course of mitral stenosis would point to the presence of tricuspid obstruction.

Another feature of considerable significance is the effect of the systolic murmur, which is usually heard in the tricuspid area on the first sound. If the murmur *follows* and does not obscure the first sound, the amount of regurgitation through the tricuspid orifice

cannot be great. If, at the same time, the signs of portal and systemic venous engorgement and of enlargement of the right side of the heart are more pronounced than the degree of insufficiency would account for, the presence of tricuspid stenosis may reasonably be suspected.

Again, since regurgitation through the tricuspid orifice tends to destroy the first sound, while stenosis tends to shorten and exaggerate it, the presence of a sharp and loud first sound, accompanied by a systolic murmur, would be evidence in favour of the presence of stenosis.

Mackenzie considers that presystolic (*i.e.* auricular systolic) pulsation of the liver suggests the presence of tricuspid stenosis. The phenomenon depends, of course, on the backflow of blood into the hepatic veins that may be produced by the auricular contraction.

ESTIMATION OF THE DEGREE OF NARROWING IN TRICUSPID STENOSIS

Since compensation to any effective degree is impossible, the estimation of the amount of stenosis has a purely scientific interest

The chief sources of information are—

1. The degree of enlargement of the right auricle
2. The extent of the systemic and portal congestion
3. The degree of arterial anæmia

CHAPTER XVII

PULMONARY INCOMPETENCE

Pathogenesis—Morbidity—Anatomy—Effects on the Heart and Circulation—Association with other Cardiac Developmental Anomalies—Symptoms—Physical Signs—Diagnosis—Estimation of the amount of Regurgitation.

ÆTIOLOGICAL PATHOLOGY

Incompetence of the pulmonic valve is an exceedingly rare condition. It occasionally occurs as a congenital malformation, when it is nearly always combined with constriction of the orifice.

According to Barié, pulmonic insufficiency, as an acquired lesion, is found most frequently between the ages of eighteen and thirty-four. The endocardial inflammation, on which the valvular incompetence depends, usually arises in connection with rheumatism, or with one of the acute infectious fevers.

In rare instances pulmonic regurgitation is the result of malignant endocarditis.

Degenerative processes, in the form of sclerosis or atheroma of the valve, are very uncommon, but they have occasionally been described in connection with both the congenital and acquired varieties of pulmonic incompetence.

There can be no doubt that relative incompetence of the pulmonic valve does sometimes occur, but the lesion is decidedly rare. It depends on the stretching of the pulmonic orifice that may be produced by high pressure in the pulmonary artery, such as, for instance, may obtain in cases of mitral stenosis.

Pulmonic incompetence due to congenital malformation of the valve, depends, as already explained, on alterations in the number, shape, or size of the cusps, which are consequently unable to efficiently close the orifice.

The morbid changes, in the acquired form of the disease, lead to thickening, induration, and distortion of the cusps, so that they are unable to come into complete apposition. In occasional instances ulceration or perforation of the cusps is the cause of the valvular insufficiency.

Incompetence of the pulmonic valve is frequently combined with constriction of its orifice.

PATHOLOGICAL RESULTS

Effects on the Heart and Circulation

The right ventricle, which is now supplied with blood from two sources, undergoes dilatation from overfilling. It subsequently hypertrophies in consequence of the additional work entailed in the propulsion of a greater quantity of blood than normal.

It is by means of dilatation and hypertrophy of the right ventricle that compensation is established in pulmonic incompetence.

Sooner or later relative incompetence of the tricuspid valve becomes established, and this is followed by dilatation and hypertrophy of the right auricle, and by engorgement of the portal and systemic veins in the manner previously described.

The diminished supply of blood to the lungs and left heart leads to a greater or less degree of arterial anæmia.

In congenital cases pulmonary incompetence is often found to be associated with other developmental anomalies, as, for instance, patency of the ductus arteriosus, or of the foramen ovale, or imperfect ventricular septum.

SYMPTOMS

So long as compensation is good, the symptoms are mainly those of derangement of the pulmonic circulation. Thus the patient usually suffers from cough and shortness of breath, especially on exertion, and these symptoms are intensified by the pulmonary complications, such as bronchitis, emphysema, or tuberculosis of the lungs, which frequently coexists with the valvular affection.

After the establishment of tricuspid regurgitation, the signs and symptoms of general venous stasis make their appearance. Death is usually due to gradual failure of the right heart.

In pulmonic incompetence due to congenital causes, the evidences of portal and systemic venous embarrassment are present to a greater or less degree from the time of birth.

PHYSICAL SIGNS

Physiognomy.—The aspect of the patient is frequently suggestive of imperfect aëration of the blood, and this is especially noticeable in cases of congenital origin. Clubbing of the fingers is also commonly found.

The signs of anæmia are sometimes very pronounced in the acquired form of the disease.

The occurrence of tricuspid regurgitation is followed by dropsy, which may be very extensive.

Pulse.—The pulse does not, as a rule, present any features of special interest. It may be irregular, both in force and frequency.

The radial artery is usually small and easily compressible, and the pulse wave short, weak, and badly sustained.

Heart.—If the tricuspid valve is incompetent, the jugular veins will be turgid and distended. They may also exhibit pulsation.

The signs of enlargement of the liver will likewise be observed.

Pulsation is occasionally seen in the intercostal spaces to the right of the sternum. Epigastric pulsation is well marked, and the area of cardiac dullness is increased chiefly towards the right.

It is sometimes possible to detect a diastolic thrill in the pulmonic area.

A diastolic murmur, partially or wholly replacing the second sound, is audible to the left of the sternum over the base of the heart. The site of maximum intensity of the murmur is situated over the second left intercostal space close to the sternum, or over the third left costal cartilage.

The murmur, which may be soft and blowing, or rough and rasping in character, is propagated downwards and to the right.

The pulmonic second sound is weak or inaudible.

DIAGNOSIS

The chief difficulty in the diagnosis of pulmonic regurgitation is the exclusion of aortic insufficiency.

The differential diagnosis of the two conditions rests on the following considerations.

1. **The pulse.**—The characteristic collapse of the pulse in aortic insufficiency is not present in the pulmonic lesion.

2. **The cardiac physical signs.**—Pulmonic regurgitation is accompanied by the signs of enlargement of the right ventricle, whereas the corresponding aortic lesion is attended by enlargement of the left ventricle.

The absence of the second sound, or the presence of a diastolic bruit over the carotid arteries in the neck, would be evidence in favour of aortic insufficiency.

The presence of great venous engorgement, with jugular or hepatic pulsation, would, on the other hand, indicate incompetence of the pulmonic rather than of the aortic valve.

Cyanosis and clubbing of the fingers would bear a like interpretation.

If the two lesions are combined a differential diagnosis may be impossible.

ESTIMATION OF THE AMOUNT OF REGURGITATION IN PULMONIC INCOMPETENCE

The degree of insufficiency is determined from the following data:—

1. **The degree of enlargement of the right ventricle.**—So long as compensation is satisfactory, the degree of enlargement of the right ventricle is a measure of the amount of regurgitation.

2. **The presence or absence of the pulmonic second sound.**—If the murmur follows and does not obscure the second sound, the amount of regurgitation is probably slight. Complete obliteration of the second sound is an indication of serious incompetence of the valve.

CHAPTER XVIII

PULMONARY STENOSIS

Pathogenesis—Morbid Anatomy—Effects on the Heart and Circulation—Symptoms—Physical Signs—Diagnosis—Estimation of the degree of Stenosis.

ÆTIOLOGICAL PATHOLOGY

WITH very rare exceptions the origin of pulmonary stenosis dates from foetal life, and the lesion constitutes one of the commonest as well as one of the most important forms of congenital disease of the heart.

Three varieties of narrowing may be observed, viz. (1) stenosis of the orifice, (2) atresia of the orifice and first part of the artery, (3) stenosis of the conus arteriosus.

In the first event the obstruction is due to intra-uterine endocarditis, which leads to adhesion of the cusps and contraction of the fibrous ring forming the orifice of the vessel. In some cases the opening is partially blocked by luxuriant vegetations, which may be formed after birth.

Atresia of the orifice and first part of the pulmonary artery is always a developmental anomaly, and is invariably associated with other malformations of the heart, such as patency of the ductus arteriosus or foramen ovale, imperfect ventricular septum, etc.

Stenosis of the conus arteriosus may be due to errors of development, but in many instances it is, no doubt, the result of myocarditis with subsequent sclerosis.

Pulmonary stenosis, as an acquired lesion, is attributable, in most instances, to endocarditis. Rheumatism and the acute infectious fevers have been the exciting cause of the endocardial inflammation in some cases. In others the lesion has appeared to depend on chronic inflammatory or atheromatous changes, the result of long-continued strain.

Direct violence has also been recorded as an exciting cause of pulmonary stenosis.

The formation of vegetations in the course of malignant endocarditis occasionally leads to obstruction at the pulmonic orifice.

PATHOLOGICAL RESULTS

Effects on the Heart and Circulation

In congenital cases the effect on the heart depends largely on the stage of cardiac development at which the pulmonary narrowing is produced. If it appears before the end of the third month, the interventricular septum does not close; if after this date, the foramen ovale and ductus arteriosus may remain patent. The condition of the right ventricle varies with the degree of occlusion of the pulmonic orifice. If the opening is completely blocked the ventricle may remain small and undeveloped, but with less degrees of narrowing the chamber is commonly greatly hypertrophied.

Since incompetence of the pulmonic valve is usually combined with stenosis of the opening, the ventricle is also found to be more or less dilated.

The cardiac changes associated with the acquired form of pulmonary obstruction resemble, for the most part, those described in connection with pulmonary incompetence.

The establishment of tricuspid regurgitation is followed by the usual train of events, culminating in portal and systemic venous engorgement, dropsy, etc. The diminished supply of blood to the lungs leads to more or less arterial anæmia.

The subjects of both the congenital and acquired forms of pulmonic obstruction are especially liable to chronic tuberculosis of the lungs, which in a large proportion of the cases is the cause of death.

SYMPTOMS

In severe cases of the congenital form of the lesion the child may not survive its birth by more than a few hours or days.

Cyanosis is usually a pronounced feature, and is accompanied by dyspnoea and the signs of general venous distension. The temperature is often sub-normal, and the fatal termination may be preceded by drowsiness, coma, or convulsions.

In less severe examples of congenital pulmonic stenosis life may be prolonged for many years, but, with few exceptions, the patient dies before the age of puberty from chronic pulmonary tuberculosis.

In cases of this kind there is usually more or less lividity of the face and extremities, especially on exertion, and the superficial veins appear distended. Clubbing of the fingers and toes is commonly observed, and the general nutrition and growth of the body is often seriously interfered with.

Dropsy does not appear as a rule until compensation fails, but it may follow urgent cardiac symptoms consequent on intercurrent pulmonary complications.

Shortness of breath on exertion, cough, palpitation of the heart, etc., are common symptoms.

The superficial temperature is commonly reduced, and the patient frequently complains of chilliness on the least exposure.

Pulmonary complications, such as bronchitis, emphysema, and tuberculosis of the lungs, are often observed, and the latter disease may be a cause of hæmoptysis.

Interference with the cerebral circulation may give rise to headache, giddiness, drowsiness, etc., and there may be impairment of the mental powers.

In the acquired form of pulmonic stenosis there may be no symptoms during the stage of compensation beyond slight dyspnoea on exertion. Clubbing of the fingers is usually present.

Failure of the right ventricle and the consequent production of tricuspid incompetence is followed by the usual signs and symptoms of portal and systemic venous congestion.

PHYSICAL SIGNS

Pulse.—The pulse of pulmonic stenosis is usually small, weak, and irregular.

Heart.—After failure of the right ventricle the jugular veins may appear distended, and in some instances they exhibit pulsation. An increase in the size of the liver is also observed.

Inspection may show bulging of the præcordium in the lower sternal region.

Epigastric pulsation is usually well marked, and is associated with a strong heaving impulse.

A thrill, systolic in time, may be felt in the pulmonic area.

The area of cardiac dulness is increased towards the right, corresponding with the enlargement of the right ventricle.

A systolic murmur, which is propagated upwards and to the left, is heard in the pulmonic area. The site of maximum intensity is situated close to the left edge of the sternum, in the second interspace, or at the level of the third costal cartilage. The murmur is not heard over the carotid arteries, but in congenital cases it may be audible over the whole præcordium.

The character of the bruit varies considerably. In some instances it is harsh and loud, in others soft and blowing.

The pulmonic second sound is weak, and may be accompanied by a diastolic murmur.

DIAGNOSIS

The site of maximum intensity, and more especially the direction of propagation of the murmur, taken in conjunction with the cardiac physical signs and the state of the arterial and venous circulations, would be sufficient to distinguish pulmonary obstruction from other valvular lesions of the heart. The exclusion of pleuro-pericardial

conditions and anæmia as the possible causes of a systolic murmur, audible in the pulmonic area, would rest on a careful consideration of the associated symptoms and physical signs.

The differential diagnosis of pulmonary stenosis and other congenital affections of the heart, such as patent ductus arteriosus, imperfect ventricular septum, etc., cannot always be made with certainty. In this respect more significance attaches to the condition of the right and left ventricles than to the site of maximum intensity and area of distribution of the murmur. Peacock states that if a case of congenital disease of the heart survive the twelfth year, the probabilities are greatly in favour of the existence of pulmonary stenosis. The differentiation between the congenital and acquired forms of pulmonary stenosis rests almost entirely on the history of the case.

ESTIMATION OF THE DEGREE OF NARROWING IN PULMONARY STENOSIS

The data which supply most information on this point are :—

1. The amount of hypertrophy of the right ventricle
2. The degree of arterial anæmia

CHAPTER XIX

COMBINED VALVULAR DISEASE

THE common combination of aortic stenosis and incompetence and of mitral stenosis and incompetence has already been pointed out. A like combination of lesions is also observed at the pulmonary and tricuspid openings.

Reference has likewise been made to the frequent association of mitral with aortic disease and of tricuspid with mitral affections.

Thus mitral stenosis may occur in combination with aortic stenosis or aortic regurgitation or with both these lesions. Again mitral incompetence may be combined with one or both forms of aortic disease, or combined mitral disease may be found in association with either or both aortic lesions. The frequent association of valvular defects on the right and left sides of the heart has already been mentioned, and the significance of the various combinations that may occur has also been considered.

The method of production of the different combinations may be inferred from what has been said on the subject of chronic valvular disease of the heart.

In all forms of combined valvular affections, however complex, each lesion is represented by its own particular physical signs.

With respect to the diagnosis of combined valvular disease, as much importance attaches to the secondary effects of each lesion on the heart and circulation as to the presence of distinctive auscultatory phenomena.

For instance, the presence of true jugular or hepatic pulsation in the course of mitral disease would justify a diagnosis of a concomitant tricuspid regurgitation, even in the absence of auscultatory evidence of the latter lesion.

The estimation of the severity of the various lesions comprising any combination is based on the considerations which were enumerated in connection with the diseases of the individual valves.

CHAPTER XX

PROGNOSIS OF CHRONIC VALVULAR DISEASE

Site and Form of Lesion—Mode of Origin and Extent of Lesion—Condition of Cardiac Muscle—State of Peripheral Vessels—General Health of Patient, etc.
—Presence or Absence of Complications.

THE considerations upon which the prognosis of chronic valvular disease of the heart is based may be conveniently arranged and discussed under the following heads:—

The Site and Form of the Lesion

The gravity of chronic valvular disease is determined to some extent by the seat and form of the affection, though this element in the prognosis is liable to so many exceptions that it does not carry great weight. Moreover, observers are not yet agreed as to the relative danger attending different varieties of valvular disease.

Provided compensation is efficient, the prognosis, at all events before middle age, is more favourable in aortic than in mitral lesions. On the other hand, if the heart fails, the prospect of recovery is better in mitral disease than in aortic. Speaking generally, the prognosis is more favourable in aortic stenosis than in aortic regurgitation, and in mitral regurgitation than in mitral stenosis. The relative gravity of left-sided valvular lesions of the heart is probably fairly accurately represented by the following order, beginning with the most serious affection, viz. (1) aortic regurgitation; (2) mitral stenosis; (3) aortic stenosis; (4) mitral regurgitation. Of right-sided lesions, pulmonary stenosis affords the most hopeful prognosis.

Tricuspid regurgitation, secondary to disease on the left side of the heart, is probably more serious than any other form of chronic valvular affection.

Aortic insufficiency is the only form of valvular disease that is liable to give rise to sudden death.

The Mode of Origin of the Lesion

Valvular rupture, which is, however, very rare, constitutes the most dangerous form of origin, and may be rapidly fatal. An acute valvulitis is less serious than chronic inflammation or atheromatous disease of the valves, since the effects in the first event are usually stationary, while in the second they are progressive. Furthermore the conditions under which chronic inflammation or atheromatous disease of the valves occur militate greatly against the establishment of adequate compensation.

As a rule the effects of an acute valvulitis are permanent and stationary, but an exception to this statement must be noted in the case of mitral stenosis, in which the narrowing tends to become more pronounced in consequence of the contraction of the newly-formed fibrous tissue. It is this circumstance which adds to the gravity of the prognosis in cases of mitral stenosis. Progressive narrowing of the opening may also occur in aortic stenosis, but the effects of the obstruction in this event are more easily neutralised than in the case of mitral constriction.

The Extent of the Lesion

The estimation of the degree of obstruction or amount of regurgitation, as the case may be, has already been fully considered under the account of the diseases of the different valves. The extent of the lesion, although an important guide to the severity of the case, is a measure of the gravity of the outlook only when interpreted in the light of the other prognostic indications.

The Condition of the Cardiac Muscle

The state of the myocardium, which forms the chief element in the prognosis of chronic valvular disease, is largely computed from the evidence afforded by the absence or presence of symptoms, by the physical examination of the heart and circulation, and by the effects of treatment.

An absence of the symptoms and signs of circulatory disturbance argues a healthy condition of the myocardium, and justifies a hopeful prognosis. Manifestations of cardiac inadequacy may appear on the arterial or on the venous side of the circulation, in consequence of the imperfect propulsion, or of the damming back of blood. The relative proportions of hypertrophy and dilatation, and the strength and size of the pulse would afford further evidence of the condition of the cardiac muscle.

A history of repeated attacks of rupture of compensation, or of gradual failure of the heart without adequate cause, is of grave import. Long-continued visceral congestion, accompanied by severe

dyspnœa, dyspepsia, albuminuria, or dropsy of the extremities and serous cavities, renders the outlook very grave.

The absence of any response to treatment implies exhaustion of the cardiac muscle and warrants a most unfavourable prognosis.

The State of the Peripheral Vessels

Arterial degeneration is serious on account of the increased strain thereby thrown on the heart. Moreover, rupture of the diseased wall may be followed by fatal hæmorrhage into the brain or elsewhere.

Atheromatous disease of the coronary arteries may directly interfere with the nutrition of the heart by impeding the circulation of blood through the organ.

Habitual high arterial tension also adds to the danger by increasing the work of the cardiac muscle.

Petechiæ are of bad omen.

The General Health of the Patient

The state of general nutrition, the presence or absence of anæmia, and the condition of the assimilative and excretory organs are the points which require special attention.

The absence of anæmia, and the existence of a good standard of general health are favourable signs.

Persistent congestion of the organs of assimilation and excretion is followed by more or less deterioration in the quality of the blood which seriously interferes with the nutrition of the heart and other viscera, and in this way materially adds to the gravity of the outlook.

The Age, Sex, Mode of Life, and Habits of the Patient

The prognosis of chronic valvular disease is less favourable in childhood than during adult life, on account of the liability to acute intercurrent disease, and of the additional strain to which the heart is exposed at the time of puberty, more especially in the case of girls. Furthermore, the special liability of the female sex to mitral stenosis and to anæmia, renders the outlook more serious in girls than in boys.

Again, compensation in cases of valvular disease occurring after the age of forty is hardly ever satisfactory, and this fact, taken in conjunction with the tendency to arterial and myocardial degeneration after this period of life, is of considerable importance in the estimation of the probable course and duration of the lesion.

Exposure, insufficient food, laborious occupations, etc., add greatly to the gravity of valvular disease, consequently the prognosis is much less favourable among the poorer classes than among the well-to-do.

Mental strain and excitement, and over-indulgence in the use of

alcohol, tea, coffee, food of all kinds, and tobacco, exercise a prejudicial effect on the course of the disease.

Temperament appears to have a considerable influence for good or evil; at all events, an even, happy disposition exerts a most beneficial effect on the production and maintenance of compensation.

Hereditary tendencies carry very great weight in the estimation of the probable duration of life in cases of chronic valvular disease, inasmuch as the liability to the premature development of arterial and myocardial degeneration is prone to run in families.

The Presence or Absence of Complications

The presence of complications, more especially affections of the lungs and of the organs which subserve assimilation and excretion, add greatly to the gravity of the prognosis. Even in the absence of complications, an accident such as cerebral embolism may lead quickly to a fatal termination, and thus upset an otherwise favourable prognosis. Except, however, in cases of aortic regurgitation, chronic valvular disease of the heart seldom gives rise to sudden death.

It goes without saying that the gravity of valvular disease is increased by the presence of pericardial, myocardial, or endocardial complications.

Pericardial adhesion, if extensive, exercises a particularly baneful influence on the establishment and maintenance of compensation.

Recurrent attacks of rheumatism add materially to the gravity of the outlook in cases of chronic valvular disease.

CHAPTER XXI

THE TREATMENT OF CHRONIC VALVULAR DISEASE

Prophylaxis—Treatment before Failure of Compensation—Treatment after Failure of Compensation—Removal of Cause—Treatment of Effects—Use of Cardiac Stimulants and Tonics.

THE prophylactic treatment of chronic valvular lesions of the heart hardly calls for comment, since the incidence of endocardial disease, whether of rheumatic or degenerative origin, cannot be prevented by any means with which we are at present acquainted.

At the same time much may be done to delay the appearance and retard the progress of chronic valvular changes, more especially when these are dependent on cardiac and vascular overstrain, such as obtains in cases of long-continued high arterial tension, or prolonged muscular exertion. It is possible, too, that the influence of gout, syphilis, and chronic alcoholism, etc., in the production of atheromatous disease of the heart and vessels may to some extent be controlled by treatment.

In practice, however, since valvular affections of the heart, with very few exceptions, are neither preventable nor curable, the main objects of treatment are, on the one hand, to promote and maintain changes of a compensatory nature, and, on the other, to ward off, retard, and minimize the effects of the lesion, in so far as they act injuriously on the heart and circulation.

In considering this part of the subject, the periods before and after failure of compensation will for convenience of description be taken separately.

Treatment before Failure of Compensation

From a clinical point of view the essential conditions for the production of compensation in cases of valvular disease following acute endocarditis are time and rest. The period that must elapse before exercise can be resumed without detriment to the compensatory process varies by weeks, or even months, and depends,

for the most part, on the site and extent of the lesion. Other things being equal, the time required for the establishment of compensation is longer in the case of aortic than of mitral disease, and in that of severe, than of slight affections.

The age of the patient, at the time of the occurrence of the lesion, also exerts a most important influence on the development of compensation, for the reason that hypertrophy of the heart originating after forty is seldom or never satisfactory. When it happens that the endocardial affection is chronic from the beginning, the compensatory changes frequently keep pace with the progress of the valvular defect.

Again, for the reasons already indicated (see p. 232), compensation is hardly ever satisfactory in those cases of *morbus cordis* which become established before the age of puberty.

In any event, so soon as compensation has been affected, the chief aim of treatment is the maintenance of the nutrition of the cardiac muscle.

This object can usually be satisfactorily attained by the intelligent observance of certain hygienic and dietetic rules, the operation of which may be supplemented, when necessary, by medicinal means.

Hygienic Treatment.—The patient may be permitted to follow his ordinary occupation and habits, provided they are not detrimental to health, and do not impose any great or sudden strain on the heart.

Exercise in the fresh air is essential, and should be encouraged in every way, but the amount that may be indulged in with safety varies greatly in different cases. For instance, a young and vigorous adult may take part with benefit in the less arduous outdoor recreations, such as cricket, tennis, golf, or cycling, etc., so long as he does not experience any unusual shortness of breath or palpitation while so engaged. On the other hand, patients of middle age, and more especially those of sedentary habits, must observe much greater caution in the amount of exertion that they undertake.

Exercise, under any circumstances, must be commenced gradually, and increased slowly and judiciously up to the maximal limit consistent with safety, which for practical purposes is defined by the onset of breathlessness, palpitation, a feeling of fatigue or of faintness.

Sudden or violent effort ought at all times to be carefully avoided.

The patient will derive benefit from cheerful surroundings, and he should be removed so far as practicable from all sources of mental strain and excitement, whether pleasurable or otherwise.

The risk of exposure to cold, damp, and sudden changes of temperature may be lessened by the adoption of woollen or flannel underclothing, which should be worn next to the skin all the year round. Ordinary common-sense precautions will be sufficient to guard against the danger of chill, and the patient should be impressed with the necessity of observing a reasonable amount of care in this respect.

A regular daily action of the bowels is of the utmost importance,

and if this cannot be obtained naturally, it must be procured by medicinal means. A tendency to constipation may be met by a course of mild saline aperients, which is usually all that is required.

Many cases of compensated cardiac disease derive great benefit from a visit to the seaside, while others appear to do better at inland places situated at an elevation of one to two thousand feet, where the climate is mild, dry, and moderately bracing.

If it is considered advisable to communicate to the patient the nature of his complaint, the information should be conveyed with the utmost caution and gentleness, and at the same time he should be encouraged to take a cheerful and sanguine view of his condition. The fear of sudden death, which in the large majority of cases is totally groundless, must so far as possible be allayed.

Dietetic treatment.—There is no necessity for any particular modification of the diet during the stage of compensation. The patient may take the food to which he is accustomed so long as it agrees with him and is moderate in quantity.

It is very desirable, however, that the total quantity of food consumed in the twenty-four hours should be fairly evenly distributed over the three principal meals of the day, in order to avoid overloading the stomach at any one repast. The digestion of a heavy meal is in itself a source of considerable circulatory disturbance and may, by this means, throw an injurious amount of strain on the heart. Furthermore, the ingestion of a large quantity of food at one time must give rise to more or less distension of the stomach, which, by direct pressure, may seriously interfere with cardiac action. It is in this way that flatulence may be a source of discomfort or even of danger.

Alcohol, whether in the form of wine, beer, or spirits, must be taken in very small quantities and at meal times only. The existence of high arterial tension is an indication for the total prohibition of alcoholic beverages, as well as for considerable restriction in the amount of nitrogenous food.

Tea and coffee are injurious unless taken in strict moderation. Tobacco must be used sparingly, on account of its depressing influence on the heart when smoked to excess.

Medicinal treatment.—The use of drugs is seldom called for at this period of the disease, since the general health of the patient and the nutrition of the cardiac muscle are usually perfectly well maintained by the measures above indicated.

Nevertheless, a liability to the appearance of anæmia is sometimes observed even under the most favourable hygienic and dietetic conditions, and it then becomes necessary to give hæmatinics, such as iron and arsenic, with which may be combined small doses of digitalis or strychnine.

Iodide of potassium with arsenic or iron make a very suitable

combination in the treatment of chronic valvular affections, and these drugs are more particularly indicated in those cases in which syphilis is a causal factor.

The following are illustrative prescriptions :—

℞	Tincturæ Ferri Perchloridi	...	℥ x.
	Liquoris Strychninæ Hydrochloridi	...	℥ iii.
	Tincturæ Digitalis	℥ viii.
	Syrupi Limonis	...	℥ i.
	Glycerini	℥ i.
	Aquæ q.s.	ad ℥ i.

Fiat Mistura. S. Two tablespoonfuls to be taken three times a day after meals.

Or,

℞	Ferri et Ammonii Citratis	...	gr. x.
	Tincturæ Nucis Vomicae	...	℥ viii.
	Liquoris Arsenicalis	...	℥ iii.
	Syrupi Aurantii	...	℥ ss.
	Aquæ Chloroformi	... q.s.	ad ℥ i.

Fiat Mistura. S. Two tablespoonfuls to be taken three times a day after food.

Or,

℞	Ferri et Ammonii Citratis	...	gr. x.
	Potassii Iodidi	...	gr. iv.
	Liquoris Arsenicalis	...	℥ iii.
	Spiritus Chloroformi	...	℥ x.
	Aquæ Menthae Piperitæ	q.s.	ad ℥ i.

Fiat Mistura. S. Two tablespoonfuls to be taken three times a day after food.

Medicinal treatment is, however, more commonly required at this stage in order to combat the effects of intercurrent disease, notably attacks of rheumatism or bronchial catarrh, which may seriously imperil the continuance of compensation.

The incidence of complications of this kind necessitate the free administration of cardiac tonics and stimulants so long as there is any danger of failure of compensation, in addition to the vigorous treatment of the concomitant affection.

To sum up, the establishment and maintenance of compensation is most advantageously secured through the promotion of the general health by means of the intelligent application of the various hygienic, dietetic and medicinal measures that have just been enumerated. It is, however, a great mistake to suppose that the best results in chronic valvular disease are obtained by the rigid application of "hard and fast" rules of treatment; each case must be considered on its merits. In attempting to steer between the Scylla of cause, and the Charybdis of effects the physician must turn to account all those conditions of constitution, habits, and temperament which operate favourably, and ward off, counteract or minimize those influences which act prejudicially on the health of the patient in general, and on the heart and vessels in particular. Thus the repression of excesses does not mean the cultivation of asceticism

the avoidance of fatigue and exhaustion does not entail the adoption of physical and mental inactivity and slothfulness; it is between these extremes, into the paths of moderation, that the lives of those suffering from chronic valvular disease must be directed, and it is only by an intelligent consideration of each particular case in all its bearings, that this result can be attained.

Treatment after Failure of Compensation.

The circulatory equilibrium established by the most perfect compensation, though stable for ordinary requirements, within strictly moderate limits, cannot be maintained in the face of sudden or prolonged effort.

Moreover, the existence of compensation carries with it certain difficulties and dangers, which are partly nutritional, and partly the result of strain on the heart and vessels. These forces, assisted by the particular tendency to interference with the circulation, both systemic and pulmonic, that is associated with each form of valvular disease, are a constant and formidable menace to the maintenance of compensation.

Sooner or later it usually happens, as the result either of extrinsic or intrinsic causes, such as have been mentioned, that compensation breaks down and dilatation of the heart from failure supervenes.

During the early stages of this process the discovery and removal of the cause of the cardiac insufficiency will, with the help of the treatment hitherto employed, frequently suffice to re-establish compensation and restore the balance of the circulation.

If, however, these measures fail to afford the requisite amount of relief, or if the cause of the breakdown cannot be discovered, or is irremovable, the failure of the heart must be combated from the side of its effects.

In practice the two methods of treatment are, as a rule, employed together, and rightly so, since the one is the complement of the other; but, whenever possible, the removal of the cause of the cardiac weakness is the object to be primarily attained.

Treatment of the Cause.

Dilatation of the heart from failure in cases of compensated valvular lesion may be due to high arterial tension, physical or mental overstrain, worry or anxiety, and acute intercurrent disease, more especially rheumatism and pulmonary disorders. It is also commonly brought about by malnutrition of the myocardium, the result of insufficient or improper food, dyspepsia, anæmia, etc., and by the abuse of alcohol and tobacco.

High arterial tension can be reduced and kept within safe limits

by means of careful dieting, and the use of purgatives and other drugs that assist in the elimination of imperfectly oxidized waste products from the blood.

Nitrogenous food, in the form of meat or meat extracts, must be used in strict moderation, and should not be taken more often than once a day. Plenty of fresh air, and exercise of a suitable kind are also important *desiderata*.

A mercurial purge, consisting of small doses of calomel, blue pill, or grey powder, should be administered once or twice a week, and may be followed, after a few hours' interval, by a draught of some saline aperient, preferably the sulphate or phosphate of soda.

The salts of potash, soda, and lithia also act as eliminating agents, and may be given, as occasion demands, in conjunction with the measures already indicated.

The reduction of high arterial tension may be rapidly effected, during an emergency, by the administration of vaso-dilators, such as nitro-glycerine, nitrite of amyl, etc., or by the direct abstraction of blood.

Failure of compensation and dilatation of the heart from over-strain must be met by the removal of the cause and the substitution of complete rest so long as there are any symptoms or signs either of cardiac irritability, or of circulatory disturbance.

Exercise should be resumed with as much care as if compensation had been established for the first time.

When mental overwork, worry, or anxiety is suspected as the cause of the cardiac failure, an attempt must be made by means of change of air, scenery, and surroundings to remove or minimize the danger arising from any of these sources.

The treatment called for by the incidence of acute intercurrent disease has already been referred to in the section dealing with the maintenance of compensation.

An effusion into the pleural, pericardial, or peritoneal cavity, occurring as a complication of morbus cordis, requires early removal, since the strain imposed on the heart by the accumulation of even a small quantity of fluid in any of these situations may give rise to serious embarrassment of the organ.

Dilatation of the heart from failure consequent on malnutrition of the myocardium is commonly found among the poorer classes as the result of exposure, insufficient food, or insanitary surroundings. Compensation is, as a rule, readily restored in these cases by the rest, warmth, and wholesome diet that can be obtained in a hospital.

The nutrition of the heart can also be promoted by medicinal means. Under the circumstances, the most useful drugs for this purpose are iron and arsenic, which may be given alone or in combination with some general tonic such as quinine, strychnine, or phosphorus.

A similar plan of treatment should be adopted with respect to the dilatation of the heart that is associated with anæmia.

It is only when these measures fail to restore compensation that recourse need be had to the assistance of digitalis or other cardiac tonics.

The treatment of the malnutrition of the heart that depends on over-eating and drinking, insufficient exercise, and on excesses of various kinds must commence with the removal of the cause. If this can be effected, and the patient is prepared and able to regulate his habits and mode of life in accordance with the general principles laid down in the previous section, the restoration of compensation is usually only a matter of time.

A few weeks' complete rest in bed, in conjunction with a course of massage, forms a useful preliminary measure in the treatment of these cases. Care must be taken, when exercise is resumed, not to overtax the heart.

The various means that were previously recommended for the purpose of promoting a healthy supply of blood to the myocardium are of equal service in the treatment of the cardiac malnutrition that is due to the causes under consideration.

Considerable benefit may also be derived in cases of this kind from the method of treatment devised by Dr. Schott, of Nauheim, which consists in the use of still or ærated baths, and carefully graduated resisted muscular movements. The effect of the baths and exercises on the heart and circulation is similar, and stated briefly is a slowing of the pulse rate with an increase in the volume and force of the beat, and a decrease in the area of cardiac dulness, *i.e.*, an improved circulation with a diminution in the size of the heart.

The physiological interpretation of these results, though still subject to dispute, is, in all probability, that the baths and resisted exercises give rise to dilatation of the arterioles of the skin and muscles respectively, with a consequent reduction in peripheral resistance and a fall of mean arterial blood pressure. The widening of the arteriolar bed in the skin and muscles, as the case may be, drains the blood from the venous to the arterial system, and more particularly from the visceral to the systemic circulation. The heart is relieved, therefore, both on the arterial and on the venous side of the circulation, inasmuch as the organ is contracting against diminished resistance with a decreased load. The fall in the pulse rate is not in the opinion of the author a serious objection to this view, having regard to the condition of the heart and circulation which commonly obtains when the treatment in question is employed.

Speaking generally the subjects of mitral disease deserve greater benefit from baths and resisted movements than those suffering from aortic lesions. In myocardial affections the treatment is sometimes of very great service. Widespread arterio-sclerosis and

advanced cardiac failure are contra-indications to the employment of this form of treatment.

Apart from the method that has just been considered, the employment of massage baths and needle baths, with water of varying temperatures, is often of very great service in the treatment of failure of compensation in chronic valvular disease.

Treatment of the Effects

The disturbance of the circulation that accompanies failure of compensation and dilatation of the heart is directly or indirectly the outcome of the defective driving power of the ventricles, and appears on the one hand as arterial anæmia, and on the other as venous congestion, together with their effects.

The attempt to relieve these conditions and to restore compensation may be made in the first instance from the side of the circulation that stands in the greater need of assistance.

Thus it may happen, as, for instance, in cases of aortic regurgitation when asystole threatens, that an immediate increase in the power of the heart is urgently required; and in such an event free stimulation of the organ offers the only prospect of relief, and must be employed irrespectively of any other consideration. On the other hand, venous engorgement and distension of the right side of the heart may become so great that failure of the circulation is an imminent danger, and in this case assistance can be rendered only by depletive measures.

As a general rule, it is preferable to reduce cardiac distension and venous engorgement, and diminish the work of the heart before increasing the force of its contractions. This method of procedure has, moreover, many practical advantages, and it will be adopted in the following account of treatment.

The Reduction of the Work performed by the Heart in conjunction with the Relief of Venous Congestion and of the Morbid Conditions associated with it.

Rest, more particularly complete rest in bed, constitutes one of the most available and powerful means of reducing the work of the heart, and there can be no doubt of its great value in the treatment of valvular disease after the occurrence of failure of compensation. At the same time, the rigid enforcement of complete rest must be tempered by tact and judgment, since strict confinement to bed is found intolerably irksome by some patients, whereas a little relaxation in the observance of the procedure will often enable long periods of inactivity to be borne with comparative equanimity.

Warmth is also an important element in the treatment, for by relaxing the cutaneous vessels it facilitates the onward flow of blood, and thus diminishes the work of the heart. Cold, on the other hand, by contracting the superficial arterioles, adds to the difficulties of the circulation.

The first question to be decided in the treatment of cardiac and venous engorgement is whether the removal of blood is necessary for its relief. If the distension of the right side of the heart is so great that failure of the ventricle is threatened, there can be no hesitation in the abstraction of blood, either by venesection or by leeches. Venesection should, however, not be employed unless there is evidence that the ventricular muscle is sufficiently powerful to be able to respond to this sudden mode of relief. It is not, as a rule, necessary to remove more than eight or ten ounces of blood, and this can be most conveniently taken from one of the superficial veins of the arm.

In less severe cases, as well as in those instances in which venesection is inadvisable, relief can usually be obtained by the use of leeches, eight or ten of which may be applied over the præcordial, or preferably the hepatic region.

Irrespective of the employment of these measures, the treatment of cardiac and venous engorgement consists in the administration of purgatives and diuretics, in conjunction with cardiac stimulants and tonics.

The bowels should be kept freely open, at first by means of calomel (gr. i-iii) and compound jalap powder (gr. xx-xxx), and later on by small doses (gr. i-iii) of calomel, blue pill, or grey powder, with rhubarb or colocynth, which may be given at night two or three times a week and followed next morning by a full dose of a saline aperient.

The beneficial effect of free purgation on the engorgement of the portal and systemic venous circulations is greatly increased by the diuresis that so commonly follows the relief of renal congestion. The action of the kidneys can be still further assisted by digitalis and other cardiac tonics which, in view of the reduction that has been effected in the amount of work that is required of the heart, may now be employed with advantage. The combination of digitalis, squill, and blue pill, or of digitalis and caffeine, is sometimes of great service in promoting diuresis. The salts of potassium, spirit of nitrous ether, etc., may be used for the same purpose.

It occasionally happens that the exhibition of diuretin will promote diuresis after the failure of all other remedies. The drug should be given in ten or fifteen grain doses every three or four hours.

It is sometimes necessary, while relieving the heart of work, to obtain a temporary increase in the force of the cardiac action, and this may be done by the use of stimulants, such as alcohol, ether, ammonia, or strychnine. On the other hand, a transient decrease in

the work of the organ may be effected during an emergency by means of nitroglycerine and the nitrites of amyl and sodium, etc., which, by dilating the arterioles, lessen peripheral resistance.

The treatment of the more important symptoms and morbid conditions that may be associated with venous congestion will now be briefly considered.

The relief of the dyspnoea, which depends on engorgement of the pulmonary circulation, can be obtained only by the measures that have hitherto been employed in the treatment of venous congestion. When the shortness of breath is aggravated by bronchitis, expectorants, such as the carbonate of ammonia, squills, senega, etc., should be added to the other remedies.

The inhalation of oxygen is also of service by relieving the insufficient aëration of the blood.

Paroxysmal attacks of dyspnoea, which are frequently associated with high arterial tension, must be treated by free stimulation of the heart and the administration of nitrite of amyl or nitroglycerine.

Severe and continuous dyspnoea, which cannot be relieved by the ordinary methods of treatment, is sometimes greatly benefited by the hypodermic injection of small doses (gr. $\frac{1}{8}$ to $\frac{1}{4}$) of morphia.

Congestion of the portal circulation is usually accompanied by more or less disturbance of digestion; consequently the regulation of the diet is a matter of the first importance. There is no objection to solid food so long as the patient can digest it, but sooner or later it becomes necessary to give nourishment wholly or partially in a liquid form.

The treatment of dropsy is identical with that of venous engorgement, and consists therefore, in the use of diuretics and hydragogue cathartics. If these remedies fail to afford sufficient relief, the oedematous tissues may be drained by puncture of the skin, which should be done, under strict antiseptic precautions, by means of Southey's trochars.

Effusion into any of the large serous cavities may be dealt with by paracentesis, and should in all cases be dealt with promptly.

Insomnia, though it is not necessarily a consequence of venous congestion, frequently accompanies the disturbance of the cerebral circulation that is associated with failure of the heart. The sleeplessness is sometimes obviously due to an alteration in the state of arterial tension, but as a general rule the precise circulatory defect upon which it depends is obscure.

When insomnia is due to high arterial tension it can be readily relieved, as Sir William Broadbent has shown, by a mild mercurial aperient, which should be given an hour or two before sleep is desired.

The sleeplessness that depends on low systemic tension must be treated by stimulants and cardio-vascular tonics. A dose of caffeine at night will often produce sleep in cases of this kind.

Hypnotics, of which sulphonal, bromide of ammonium, and paraldehyde are the most useful, should always be used with caution.

Massage is frequently of service in the treatment of the insomnia that is due to circulatory causes.

When other means fail, sleep can often be obtained by the subcutaneous injection of a small dose (gr. $\frac{1}{6}$) of morphia.

The Increase in the Power of the Heart

Stimulation of the heart by means of alcohol, ether, the salts of ammonium, or strychnine is the most rapid and powerful method of temporarily increasing the force of its contractions. In conditions of great urgency these remedies can be administered subcutaneously. Under less pressing circumstances half-drachm doses of ether and sal volatile, with two or three minims of liquor strychninæ, may be given by the mouth every two, four, or six hours, as occasion requires.

Alcohol, in the form of spirits, is a very convenient and powerful cardiac stimulant, and it possesses the additional advantage of mixing well with most of the ordinary liquid foods. The dose must be regulated in accordance with the degree of stimulation that is required. It is seldom, if ever, necessary to prescribe a daily allowance of more than ten or twelve ounces of alcohol, which should be taken in small quantities, at regular intervals, during the twenty-four hours.

The application of hot turpentine stupes, mustard poultices, or a few small fly blisters to the præcordium, and of pungent substances to the nose, are also of service in stimulating cardiac action, and they may be employed with the other measures that have been mentioned. An increase in the force of the heart of a more durable quality can be obtained by the administration of the so-called "cardiac tonics," of which digitalis is by far the most useful, though other members of the group, notably strophanthus, caffeine, squill, convallaria, sparteine, etc., are sometimes of very great service.

These remedies may be given with ether, the salts of ammonium, or strychnine, or with iron, arsenic, nux vomica, phosphorus, quinine, etc., according to the special needs of the heart and the general requirements of the patient. The action of the cardiac tonics is frequently more effective when two or three are used together than when the same drugs are employed separately and successively, as may be observed with combinations of digitalis and caffeine or squill, and of strophanthus and convallaria majalis. Moreover, this method of administration is in all probability capable of considerable extension.

A purge should in all cases precede and from time to time co-operate with the use of cardiac tonics.

The physiological action of digitalis, which will be taken as the type of the remedies under consideration, is exerted for the most

part on the muscular tissue of the heart and arterioles. Its effect, in moderate doses, is to increase the force and reduce the frequency of the cardiac beats, and at the same time to raise the tension in the arteries.

The indications, therefore, for the therapeutic use of digitalis are failure of the heart with dilatation of its cavities, a rapid, weak, and irregular pulse, venous engorgement, dropsy, and congestion of the viscera, as shown by dyspnoea and a scanty secretion of urine.

Increased functional activity of the kidneys, a diminution of the dropsy, and a slower, stronger, and more regular pulse are the most reliable manifestations of the beneficial action of the drug.

Strophanthus is a useful substitute for digitalis when the latter drug cannot be tolerated or has failed to do good. The precise conditions for the successful employment of this remedy have not yet, however, been fully ascertained.

The action of *convallaria majalis* appears to be very uncertain, but the drug is sometimes of service, more especially when given in combination with strophanthus.

Sparteine produces a prompt but temporary increase in the power of the heart, consequently it may be used with strychnine or other cardiac stimulants in order to tide over an emergency.

The action and uses of digitalis will now be considered in greater detail.

The relief afforded by the drug in the treatment of failure of the heart and its effects is obtained by (1) the increase in the contractile power and tone of the cardiac muscle, whereby the ventricles are enabled to more completely expel their contents, while the size of their cavities becomes lessened; (2) the lengthening of the diastole of the heart so that not only is there more time for the nutrition of the organ, but also for the outflow of blood from the great veins, which is still further assisted by the improved suction power of the stronger ventricles; and (3) the rise of pressure in the arteries, which are kept better filled by the more vigorous ventricles, whence the flow of blood into and through the capillaries and veins is maintained at a more rapid and regular rate.

The diuretic action of digitalis, which is often greatly assisted by the co-operation of mercury and of caffeine, is accounted for by the rise of pressure in the renal arteries and the consequent improvement in the circulation through the kidneys, together with the increase in the watery constituents of the blood that attends the absorption of dropsical effusions. The latter circumstance is the most powerful factor in the production of the diuresis; for in the absence of dropsy digitalis has but little influence on the secretion of urine.

The effects of digitalis are not equally serviceable in all forms of valvular disease. While there is no doubt that the drug is of very great value in mitral regurgitation, whether primary or secondary

to other valvular lesions, it is of much less service, and at times altogether harmful, in mitral stenosis, and in aortic incompetence and aortic obstruction.

The beneficial influence of digitalis in mitral regurgitation is explained by the increase in the amount of contraction of the left ventricle, plus the decrease in the size of its cavity, whereby the auriculo-ventricular opening is not only constricted, but is also better protected (by reason of the closer approximation of the valvular curtains), and in both these ways the leakage through the orifice is lessened. Furthermore, the improvement in the pulmonary circulation consequent on the increased vigour of the right ventricle leads to a larger and more equable supply of blood to the left auricle, and hence to the left ventricle and aorta, while the accompanying rise of intra-auricular pressure assists in resisting the reflux through the mitral opening. It is of course to its effects on the right ventricle and pulmonic circulation that the beneficial influence of digitalis in mitral regurgitation is chiefly attributable.

The initial effect of digitalis in mitral stenosis is frequently beneficial, and is due, no doubt, to the prolongation of the diastole of the heart and the greater power of the right ventricle, whence more time is allowed for the discharge of the auricular contents, while the *vis a tergo*, in the shape of heightened pulmonic tension, is increased.

The subsequent unfavourable action of the drug is explained by Sir William Broadbent, on the grounds that, so soon as the pressure in the pulmonic circulation and left auricle has reached the point at which the blood is forced through the narrowed mitral opening at the maximum rate of speed, further stimulation of the right ventricle can only give rise to embarrassment of the organ.

It may be, too, that in the absence of hypertrophy of the left ventricle, the increase of systemic arterial tension offers a greater resistance to the systole of the heart than the organ is able to cope with.

When digitalis disagrees in cases of mitral stenosis, its place may be taken by a combination of strophanthus and convallaria majalis, which frequently gives surprisingly good results. Since this combination exerts comparatively little influence on arterial tension, it seems probable that the ill effects of digitalis in mitral stenosis depend, to some extent at least, on vasomotor changes. If incompetence of the mitral valve is associated with narrowing of the opening, the effects of digitalis will be beneficial in proportion to the extent of the regurgitation.

The effects of digitalis in aortic incompetence will be harmful in so far as the amount of backflow is augmented by the rise of arterial tension and the prolongation of the diastole of the heart, and beneficial in so far as the dilatation of the ventricle is reduced by the increase in the contractile power and tone of its muscular walls.

Opinions are divided with regard to the therapeutic value of digitalis in aortic insufficiency, for while some observers look upon it as injurious, others consider it to be of great service in the treatment of this disease. Sir William Broadbent suggests that these conflicting views can be explained by the fact that the influence of digitalis in aortic regurgitation is harmful under one set of conditions and beneficial under another. Thus if the failure of compensation in aortic incompetence manifests itself predominantly in the form of arterial anæmia, digitalis does harm; whereas, if in the form of mitral backworking, with venous congestion, etc., it does good, for the reasons given under the head of "Mitral Regurgitation."

Be this as it may, there is no doubt that the plan of procedure underlying Broadbent's suggestion, forms a useful and practical working rule in the administration of digitalis in cases of aortic incompetence.

It sometimes happens that a combination of digitalis and strophanthus will succeed where the former drug alone has failed to be of service.

Aortic stenosis derives most benefit from digitalis when the failure of compensation is accompanied by evidence of backworking through the mitral opening. In the absence of mitral leakage digitalis may do harm, in that it increases the pre-existing arterio-capillary resistance and thus adds to the work of the left ventricle.

Strophanthus, though somewhat uncertain in its action is sometimes of very great service in the treatment of aortic disease. The drug may be given in combination with iron, arsenic, strychnine, etc., or with some other cardiac or general tonic.

CHAPTER XXII

DISEASES OF THE MYOCARDIUM

Classification—Section I. Hypertrophy and Dilatation of the Heart; Definition; Classification of Kinds of Cardiac Enlargement; Morbid Anatomy; Effects on other Organs; Pathogenesis; Ætiology; Symptoms; Physical Signs; Diagnosis; Prognosis; Treatment—Section II. Acute Myocarditis; Ætiology; Morbid Anatomy; Effects on the Heart; Symptoms; Physical Signs; Diagnosis; Prognosis; Treatment—Section III. Degenerative Diseases of the Myocardium; Definition; Ætiology; Morbid Anatomy; Pathogenesis; Symptoms; Physical Signs; Diagnosis; Prognosis; Treatment—Section IV. Fatty Diseases of the Heart; Fatty Infiltration; Fatty Degeneration—Section V. Fibroid Disease of the Heart—Section VI. Growths in the Heart.

THE muscular walls of the heart are liable to be affected by a variety of morbid conditions, which for descriptive purposes may be arranged as follows :—

1. HYPERTROPHY AND DILATATION
2. ACUTE MYOCARDITIS
3. DEGENERATIVE DISEASES
 - (a) *Parenchymatous, etc.*
 - (b) *Fatty*
 - (c) *Fibroid* (including cardiac aneurism and chronic myocarditis)
4. GROWTHS

This classification, which, from a pathological point of view, is far from exhaustive, includes nevertheless the greater number of affections of the myocardium that are of clinical significance.

Apart, however, from hypertrophy and dilatation of the heart, the differential diagnosis of myocardial disease is usually a matter of extreme difficulty, and the recognition of any particular lesion often rests as much on the general state of the patient and other causal indications, as on the circulatory phenomena. The reason for this is that we are at present unable clinically to distinguish

with anything like precision between the effects of different morbid conditions of the cardiac walls, since all tend to impair the muscular power of the heart and thus give rise to the signs and symptoms of insufficiency of the organ, accompanied by mechanical disturbance of the circulation.

SECTION I

HYPERTROPHY AND DILATATION OF THE HEART

The terms "hypertrophy" and "dilatation" as applied to the heart are used to distinguish certain processes whereby the organ increases in size. They may be defined as follows:—

1. **Hypertrophy**: An increase of the muscular tissue of the heart which leads to thickening of the cardiac walls and enlargement of one or more of the chambers of the organ.

2. **Dilatation**: A uniform enlargement of one or more of the cavities of the heart.

Two distinct varieties of dilatation are recognised, viz.:

1. Dilatation from the overfilling of a chamber which is compensatory in its effect.
2. Dilatation from the incomplete emptying of a chamber which is associated with failure of the muscular power of the heart.

The following varieties of enlargement of the heart are (after Dr Mitchell Bruce) distinguished as the result of the operation of one or more of these processes.

1. Simple hypertrophy in which the parietes of the heart are thickened while the cavities retain their normal size.
2. Compensatory dilatation with hypertrophy.
3. Simple hypertrophy with dilatation from failure.
4. "Compensatory dilatation with hypertrophy," with dilatation from failure.

The conditions referred to under numbers 2, 3, and 4, are also known as "eccentric hypertrophy" and "dilated hypertrophy."

In all of them the capacity of one or more of the chambers is increased, while the normal thickness of the walls is preserved or augmented.

At the same time, the relative proportions of hypertrophy and dilatation vary greatly in different cases.

5. Simple dilatation from failure, in which the capacity of one or more of the cavities is increased, while the walls are diminished in thickness.

The condition formerly described under the term "concentric hypertrophy," denoting thickened walls, with diminished size of cavities, has no real existence.

Hypertrophy, dilatation, or any of their combinations, may be either general or local; that is to say, they may involve, more or less, all of the four chambers of the heart, or they may be limited to one or two of its compartments.

The ventricles are more frequently the seat of hypertrophy than the auricles, and the left side of the heart is more often affected than the right. This order reversed expresses the liability of the cardiac cavities to undergo dilatation.

Hypertrophy and dilatation never occur as primary disorders. They are always secondary to some other affection, and the importance of this fact in the consideration of myocardial disease cannot be too strongly insisted upon.

PATHOLOGICAL ANATOMY

Hypertrophy

The heart appears to be elongated, more especially in the direction of the left apex, and the bulk and weight of the organ are greater than usual. Increase in the weight of the heart affords the most trustworthy evidence of hypertrophy that we possess. Irrespective of the variations produced by the method of removal that is adopted, the average weight of the normal heart in adult males ranges between 11 and 13 ounces, and in females between 9 and 11 ounces.

The walls of the chambers affected by hypertrophy, predominantly the ventricles, are thicker and firmer than normal, and do not collapse on section. The *musculi papillares* and *columnæ carneæ* usually participate in the muscular enlargement of the heart. Except in the case of the left ventricle, pure hypertrophy of the cardiac walls is seldom or never observed. The hypertrophied muscle fibre is firm and of a brownish red colour, becoming bright red on exposure.

The increase in bulk of the muscular tissue is chiefly due to the formation of new muscle fibres. Recent researches tend to show that the size of the individual fibres is slightly increased, otherwise the histological characters of the myocardium are unaltered.

Dilatation

In pure dilatation the outline of the heart is more rounded than normal, and the weight of the organ is never increased. The enlargement of the affected cavities is associated with thinning of their walls, which collapse on section.

The muscular tissue, which may be firm or soft, is variously altered in colour. Under the microscope the muscle fibres may present a normal appearance, but more often they are either the seat of inflammatory changes, or in some stage of pigmentary, granular, fatty, or fibroid degeneration.

Hypertrophy with Dilatation

This association, which is the one commonly found in cases of cardiac enlargement, combines the features of the two forms just described, *i.e.* the capacity of one or more of the cardiac chambers is increased, while the normal thickness of the walls is preserved or augmented. The size of the heart is always increased, while the shape of the organ depends on the relative extent of the two associated changes. The weight of the heart is greater than normal.

The muscular walls may be variously altered in colour, consistence, and structure.

ASSOCIATED PATHOLOGICAL CHANGES

The pathological conditions associated with hypertrophy, dilatation, or any of their combinations, vary with the site and nature of the primary lesion. In the large majority of cases disease of the valves of the heart, or of the pericardium, lungs, or kidneys, is the source of the cardiac enlargement. Apart, however, from the primary pathological conditions that may be found in association with cardiac enlargement, hypertrophy and dilatation give rise to certain changes in the heart and circulation which must be briefly considered.

The increased intra-cardiac pressure which must accompany hypertrophy, dilatation, and their various combinations, leads, sooner or later, to more or less thickening of the valves, chordæ tendineæ, and endocardium, on one or both sides of the heart. The arteries, both systemic and pulmonic, undergo similar changes in consequence of the additional strain to which they are exposed, and the subsequent degeneration of the thickened walls may be followed by aneurism or rupture of the affected vessels. Cardiac and venous thrombosis are sometimes observed in the later stages of dilatation of the heart, owing to the slowing of the circulation. Portions of the thrombi occasionally become detached, and the liberated fragments carried by the blood stream may give rise to embolism of the arteries of the heart, lungs, brain, spleen, kidneys, etc.

Pulmonary embolism is very commonly the result of systemic venous thrombosis.

Dilatation from failure, whether of the right or left side of the heart, leads ultimately to a further series of changes, which culminate in general venous congestion and dropsy, with a corresponding degree of arterial anæmia.

The effect of this process on the other organs of the body is firstly disturbance of function, and later the production of organic lesions, accompanied by fibrosis, pigmentation, atrophy, etc. The viscera chiefly affected are the lungs, liver, spleen, alimentary canal, kidneys, brain, and heart.

The more important pathological changes which are found in these organs may be briefly summarised as follows:—

Lungs.—Pigmentation, capillary engorgement, and dilatation, with more or less interstitial fibrosis of the lungs, constitute the chief effects of prolonged mechanical congestion. These changes, which are comprised under the term “brown induration” of the lungs, are usually associated with other morbid conditions, such as pulmonary oedema, and collapse, infarction, pneumonia, bronchitis, and emphysema.

Liver.—The liver is usually enlarged, but in cases of severe and protracted venous congestion it may become reduced in size as the result of cyanotic atrophy with cirrhotic changes.

The cells of the lobules undergo pressure atrophy, and may be observed in all stages of pigmentary and fatty degeneration. The peculiar mottling of the organ which may be seen on section, arises in consequence of the contrast afforded by the deep congestion of the central parts of the lobules, as compared with the lighter coloured peripheral portions, and has given rise to the term “nutmeg liver.” The fibrous stroma of the liver is increased, and there is usually some thickening of Glisson’s capsule.

Spleen.—The spleen is enlarged in the early stages of venous congestion, and shows on section a deep purple colour. It may subsequently become indurated and contracted. Infarction of the spleen is of common occurrence.

Stomach and Intestines.—The sub-mucous tissue is engorged with blood, and the mucous membrane usually gives the appearances of acute or chronic catarrh. Punctiform hæmorrhages are also frequently observed.

Kidneys.—The kidneys are congested and enlarged, and they sometimes show cirrhotic changes.

Infarction with the subsequent production of depressed cicatrices is not uncommonly found.

Brain.—The brain is congested, and thrombosis may occur in the cerebral veins and sinuses.

Heart.—The inter-muscular connective tissue is usually increased, sometimes greatly so, while the muscle fibres may be the seat of pigmentary, granular, or fatty changes.

ÆTIOLOGICAL PATHOLOGY

Hypertrophy

Cardiac hypertrophy is in all cases dependent on an increase in the amount of work required of the heart, and its occurrence is explained by the operation of two physiological laws, which are :—

1. That the functional activity of a muscle is increased by tension

of its fibres. Stated in other words, the force exerted by a muscle in its contraction is (up to a certain maximal limit) augmented by the resistance that it has to overcome, *i.e.* the greater the resistance the more powerful the contraction.

2. That if a muscle is the seat of increased functional activity for a considerable period of time it undergoes hypertrophy, provided it has an adequate nutritive supply.

The work done by the cardiac muscle consists in the propulsion forward, from an auricle into a ventricle, or from a ventricle into an artery, of a certain charge of blood. If the resistance to the onward flow in any of these situations be abnormally raised, or if the charge of blood be unduly augmented, the work of the heart is increased, and the muscular wall of the chamber on which the additional stress falls will, for the reason given above (law 1), contract more forcibly than usual, and if the difficulty persist, will (law 2), under favourable nutritive conditions, undergo hypertrophy.

The conditions, therefore, under which cardiac hypertrophy is most commonly observed are affections of the heart itself, such as valvular or pericardial disease, and disturbances of the peripheral circulation whereby the resistance to the onward flow of blood through the arteries and capillaries is abnormally raised.

Under favourable circumstances the degree of hypertrophy evoked by any of these lesions is proportional to the requirements of the occasion. It never exceeds this amount, though it not infrequently falls short of it.

In any event it will appear that hypertrophy of the heart does not occur as a primary disorder, but is always secondary to and dependent upon some other disease, the evil effects of which it removes or minimizes. The adequacy of the compensation afforded by hypertrophy in cases of morbus cordis, is gauged by the extent to which the normal balance of the circulation is restored. Compensation may be regarded as perfect when the circulatory equilibrium that is re-established by hypertrophy is stable within ordinary requirements, and this may be obtained under favourable circumstances.

Dilatation

It has already been stated that dilatation of the heart is of two distinct kinds, viz. that due to overfilling of a chamber which is compensatory in its effect, and that due to incomplete emptying of a chamber which is associated with failure of the muscular power of the heart. Yet, while the physiological and pathological significance of these two conditions afford a striking contrast, the mechanical processes whereby the cardiac dilatation is established in either event are practically identical.

In both cases the first step towards the production of dilatation of the heart is the overcharging and consequent distension of one

or other of the cardiac chambers with blood. The increase in capacity, which must be supplied in order to accommodate the additional quantity of blood, is obtained by the yielding of the elastic muscular parietes of the chamber involved.

Provided the causes which have given rise to this enlargement act with sufficient intensity, or over a sufficiently protracted period of time, the cardiac walls become permanently stretched, and the cavity they encircle becomes proportionally dilated.

The vital phenomena underlying the mechanical process just described are essentially different for the production of the two forms of dilatation of the heart. The mode of development of the condition in each case will now be considered in detail.

Dilatation of the heart from overfilling may be most conveniently studied in connection with the cardiac enlargement that attends aortic regurgitation.

In this disease the left ventricle during diastole receives, in addition to the normal supply from the left auricle, the amount that regurgitates through the imperfectly closed aortic opening. At the end of diastole, therefore, the contents of the ventricle exceed the normal by the amount of the aortic reflux. The accommodation of the overcharge necessitates an increase in the capacity of the ventricle, and the consequent stretching of its elastic walls. Expressed in other words, the forces concerned in the distension or stretching of the elastic ventricular walls *during diastole* are augmented by the pressure that is exerted by the backflow of blood from the aorta, and the increase in the capacity of the chamber is the expression of the additional distensile stress to which it is exposed. This condition of the ventricle, if perpetuated, becomes dilatation, which, as has been shown, is due to overfilling.

Pari passu with the development of dilatation the ventricle undergoes hypertrophy, by reason of the increased work entailed in the propulsion of a greater quantity of blood than usual. The dilatation of the left ventricle is as much a part of the compensatory arrangement as the concomitant hypertrophy; for so long as the ventricular contraction is carried through, the normal quantity of blood plus the amount of the backflow is projected into the aorta, and thus the supply to the systemic vessels and tissues undergoes no diminution.

The dilatation of the left ventricle (associated with hypertrophy) that accompanies mitral regurgitation is also the result of overfilling, and is compensatory in its effect, since it allows for the mitral reflux, and at the same time permits the discharge of an adequate supply of blood into the aorta.

The reasoning employed with respect to enlargements of the left ventricle applies with equal force to enlargements of the right ventricle under corresponding conditions.

It will thus appear that dilatation of a cardiac chamber from

overfilling is always the result of a valvular defect, and that it is invariably associated with hypertrophy.

The compensation effected is due (*a*) to the dilatation, in so far as it provides for the amount of the valvular leakage, and thus prevents any diminution in the supply of blood to the systemic vessels, and (*b*) to the hypertrophy, in so far as it enables the ventricle to complete its contraction and to expel its contents.

Dilatation of the heart from incomplete emptying may be contrasted with dilatation from overfilling, inasmuch as it is always the result of failure, either absolute or relative, of the cardiac walls, and consequently entails some degree of disturbance of the circulation.

The mode of development of dilatation of a cardiac chamber from incomplete emptying depends on the co-operation of forces that are partly mechanical and partly vital, and it is the introduction of the later ætiological factor which renders the origin of the condition one of considerable complexity.

In order to illustrate the subject it will be convenient to treat of the left ventricle only, but it must be understood that the reasoning employed is of general application as regards the other chambers of the heart.

Experimental research has shown that at the end of the ventricular systole a certain quantity of blood remains in the upper part of the chamber between the valves and the papillary muscles.

Any increase in the resistance to the discharge of the contents of the ventricle, as for instance a rise of peripheral tension, adds to the amount of the residual blood, which can, no doubt, vary considerably within the physiological limits prescribed by the degree of reserve power possessed by the heart. So long as this restriction is not overstepped, the resulting distension of the ventricle, necessitated by the accommodation of the normal charge from the auricle, plus the residual excess of blood, may be termed physiological.

If, however, the rise of peripheral tension persist, or be raised above physiological limits, or if the muscular power of the heart, either relatively or absolutely, be insufficient for the adequate expulsion of the ventricular contents, a time arrives when the contractile energy of the cardiac walls becomes exhausted before the completion of the systole. A certain quantity of blood, therefore, which ought to have been propelled into the aorta remains behind in the chamber, and to this is immediately added, for the time at least, the normal charge from the left auricle. Hence at the end of diastole the ventricle is overcharged by the amount of the residual excess of blood left by the preceding incomplete systole. The accommodation of this overcharge necessitates an increase in the capacity of the ventricle and the consequent stretching of its elastic walls. Moreover, with each succeeding cardiac cycle the ventricle becomes further overloaded, and unless

the cause of the distension is removed the chamber becomes permanently enlarged, or, in other words, dilated.

If the causes persist, dilatation once established tends for obvious reasons to increase; but it must not be supposed that the yielding of the cardiac walls is gradual and continuous. In response to the additional demands made upon it, the ventricle from time to time, by means of unusually powerful contractions, succeeds in temporarily getting rid of the accumulation of blood in its cavity; but the process of overloading is repeated, and step by step, under the influence of recurring strain, the amount of dilatation becomes greater. In advanced cases of dilatation from failure the ventricle is always more or less full of blood, and its inflow and output are reduced to very small dimensions.

Under circumstances of this kind the slowing of the circulation through the heart favours the clotting of blood in its cavities, and the consequent formation of emboli which may be carried to distant organs.

The production of dilatation from failure depends most commonly on the co-operation, in varying degrees, of two main factors, either of which may, however, occasionally act alone. They are (1) an increase in the work required of the heart, consequent on augmented arterio-capillary resistance, valvular disease, overstrain, etc., and (2) impairment of the muscular power of the organ, the result of structural disease of the cardiac walls, or of nutritive, neuro-muscular, and other disturbances.

Among the chief morbid conditions of the myocardium may be mentioned myocarditis, fatty and fibroid disease, and the granular degeneration of the muscle fibres that accompanies many of the acute specific fevers.

Nutritive disturbance of the cardiac walls and of their neuro-muscular apparatus may be the result of disease of the coronary arteries, rheumatism, toxæmias of various kinds, anæmia, dyspepsia, and of acute and chronic pulmonary disorders. It may also be due to a large number of other causes of a less defined influence, but none the less operative, such as emotional excitement of any kind, anxiety, grief, worry, and nervous overstrain, more particularly in young people. It is possible, too, that dilatation of the heart may, in some instances, be the direct effect on the myocardium of poisons circulating in the blood.

The abuse of tea, alcohol, and tobacco, dietetic and sexual excesses, sedentary and irregular habits, chronic constipation, etc., are also contributory factors in the production of cardiac dilatation.

Acute dilatation of the heart from failure is sometimes observed. Thus it may occur at the onset of acute renal disease, especially when this disorder is associated with scarlet fever. Here the increased arterio-capillary resistance, combined with the myocardial degeneration, which is so commonly found, leads in some instances to rapid dilatation of the left ventricle.

Acute dilatation of the heart may also be observed in the course

of many of the acute specific fevers, such as diphtheria, measles, enteric fever, influenza, etc. It may likewise be due to sudden or excessive muscular exertion, and to poisoning by alcohol and other drugs.

Acute dilatation of the right side of the heart is seen in cases of whooping cough, pulmonary embolism, over-exertion, etc.

Dilatation from failure may be grafted on to previous enlargement of the heart, *i.e.* on to hypertrophy, or on to dilatation from overfilling with hypertrophy, but it may occur without any antecedent increase in the size of the organ, and the condition is then termed primary dilatation. Any or all of the cardiac chambers may be the seat of dilatation from failure.

The process usually commences in one compartment, and gradually spreads backwards, chamber by chamber, to the others; so that sooner or later, as the case may be, the left or right side of the heart, or the whole organ, becomes involved in the morbid change. The mechanism by which this series of effects is produced has already been described under the head of "Mitral Regurgitation," and need be but briefly referred to here.

Stated shortly, dilatation of the left ventricle from failure leads to incompetence of the mitral valve, by reason of the inability of the stretched and weakened muscle fibres, surrounding the auriculo-ventricular orifice, to diminish the size of the opening sufficiently to enable the valvular curtains to come into adequate apposition.

Regurgitation through the mitral orifice is followed by dilatation of the left auricle and a general rise of pressure throughout the pulmonary circulation. The right ventricle subsequently fails before the stress of driving the blood through the lungs, with consecutive dilatation of the tricuspid opening and incompetence of its valve. Tricuspid regurgitation is followed by dilatation of the right auricle and distension of the veins of the systemic and portal circulations.

The further effects of this process of "backworking" on the heart and circulation and on the other organs of the body have been sufficiently described elsewhere.

The different forms of cardiac enlargement that have been described will now be briefly considered with reference to the various ways in which they may be combined and associated.

Hypertrophy of the heart seldom occurs in a pure form, but it is sometimes seen in cases of aortic and renal disease.

Hypertrophy of the heart, with dilatation or eccentric hypertrophy, as it is often styled, is the condition most commonly found.

Dilatation of the heart from overfilling is most typically exemplified in cases of aortic regurgitation, and is always associated with hypertrophy.

Dilatation of the heart from failure may supervene on either hypertrophy or on dilatation from overfilling with hypertrophy, or it may be observed without any previous enlargement of the organ.

The effect of hypertrophy, and of dilatation from overfilling with

hypertrophy, is always compensatory, since both these conditions counteract, minimize, or remove pre-existing cardiac disability.

Dilatation from failure is invariably the result of inadequacy, either absolute or relative, of the muscular power of the heart, and entails some degree of disturbance of the circulation.

All or any of the cardiac chambers may be the seat of enlargement. As a rule the process begins in one compartment, and spreads backwards to the others, so that sooner or later the whole heart is involved. It is by virtue of this backward distribution of the effects of heart disease that either ventricle is enabled to come to the help of its fellow.

Thus in left-side lesions of the heart the backward pressure through the mitral opening, left auricle, and lungs is met by hypertrophy of the right ventricle, which by maintaining the pulmonary circulation ensures an adequate supply of blood to the left auricle, and hence to the left ventricle and aorta. In like manner the left ventricle, through the systemic circuit, comes to the assistance of an overtaxed right heart.

The hypertrophy of the left ventricle which may accompany right-sided lesions of the heart is, in part, also the result of increased arterio-capillary resistance consequent on the reflex contraction of the small arteries through the vaso-motor centre, and on the impediment offered by the arterioles and capillaries to the circulation through them of venous blood.

ÆTIOLOGY

It will be convenient under this head to consider the causes of enlargement of each individual cavity of the heart in detail.

LEFT VENTRICLE

Hypertrophy

1. Diseases of valves.—The valvular affections which induce hypertrophy of the left ventricle are aortic stenosis, aortic incompetence, and mitral incompetence.

In aortic stenosis the hypertrophy of the ventricle, under favourable circumstances, occurs in a pure form, *i.e.* it is not associated with dilatation. In aortic and mitral incompetence the hypertrophy of the ventricle is combined with dilatation from overfilling, so that in both instances the condition known as eccentric hypertrophy is produced.

2. Diseases of the aorta and its branches.—The affections included under this heading are atheroma, congenital stenosis of the aorta, and aneurismal dilatation of the vessel.

The impairment of elasticity, and consequently the relatively rigid condition of the walls of the aorta and systemic arteries, with or without narrowing of their lumen, that is occasioned by atheroma, not only offers an obstruction to the blood current, but also directly

impedes the discharge of the contents of the ventricle, which, therefore, undergoes hypertrophy from increased work.

The nutritive conditions associated with the circumstances under which atheroma is observed are, however, hardly ever satisfactory, hence it usually happens that the ventricular hypertrophy is combined with more or less dilatation from failure.

As the result of a congenital malformation, the aorta is sometimes found constricted just beyond the point of entry of the ductus arteriosus. In this event the work of the ventricle is increased, and it consequently becomes hypertrophied.

Aneurism of the aorta may or may not give rise to hypertrophy of the left ventricle. If the aneurism involves the aortic semilunar valve hypertrophy of the ventricle is the rule, whereas it is the exception if the valve is not implicated. The ventricular hypertrophy is associated with dilatation from overfilling, to which there is frequently added dilatation from failure.

3. High arterial tension.—Next to valvular disease, the most prolific source of hypertrophy of the left ventricle is protracted high arterial tension. This condition is found most commonly in association with acute and chronic Bright's disease, especially contracted granular kidney, gout, lead poisoning, pregnancy, and arterial degeneration. It is also observed in some cases of diabetes, anæmia, and emphysema, and not infrequently in connection with cardiac neuroses and overstrain of the heart. In some instances an hereditary tendency to high arterial tension has been noticed, and in any event the liability to develop the condition becomes more pronounced with advancing years.

Among the more important predisposing causes of high arterial tension may be mentioned the habitual abuse of alcohol, the over- ingestion of nitrogenous food, constipation, and sedentary habits.

The obstruction to the free passage of blood through the small arteries and capillaries, which is the most common and important factor in the production of high arterial tension, necessitates an increased display of force by the left ventricle in the discharge of its contents, so that, provided it enjoys sufficient nutrition, the chamber will in course of time become hypertrophied.

The hypertrophy of the left ventricle due to high arterial tension is observed in its purest form in cases of contracted granular kidney. In many cases, however, the vigour of the nutritional forces is insufficient for the maintenance of adequate hypertrophy, and dilatation from failure becomes superadded.

4. Increased functional activity from prolonged muscular exertion or from nervous excitation.—Hypertrophy of the left ventricle is frequently found in persons whose mode of life entails severe and protracted muscular exertion. Thus it occurs among athletes, hammer men, hill climbers, stonemasons, and among those accustomed to carry heavy loads.

In such instances, the cause of the hypertrophy which is physiological, is twofold, viz. (1) an acceleration in the rate of the cardiac beat, and (2) an increase in the peripheral systemic tension, whereby the work of the heart is increased.

In cases of sudden, violent, and prolonged muscular effort, the limits of the heart's reserve power may be exceeded, and dilatation from failure results, with the subsequent production of hypertrophy, which may or may not be sufficient to restore the balance of the circulation.

Acceleration of the heart's beat does not necessarily imply increased work, and it is for this reason that the rapidly beating heart, due to nervous excitation, does not undergo hypertrophy, unless associated with a systemic peripheral tension, which is above the normal, or with some other condition, capable of giving rise to obstruction to the blood current.

A combination of muscular exertion and nervous excitation is the most probable cause of the cardiac enlargement known under the name of "irritable heart." This condition has been most commonly observed among soldiers on active service, a mode of life which entails much nervous strain and excitement, together with severe, sudden, and often protracted muscular exertion. The enlargement of the heart in cases of this kind, though commonly of general distribution, affects most markedly the left ventricle, which shows dilatation in association with a variable degree of hypertrophy.

The mechanism of production is in all probability an initial dilatation from failure and the subsequent development of hypertrophy, which, owing apparently to an inherent lack of cardiac reserve force, and of nutritional vigour, is insufficient to completely counteract the effects of dilatation.

Dilatation of the heart, it may be pointed out, must always be an indirect cause of hypertrophy, since the propulsion of the additional quantity of blood contained in the enlarged cavities of the organ necessitates an increased display of force.

It is possible, indeed probable, that a preliminary distension or even dilatation of a cavity is in all cases the immediate precursor of hypertrophy of its walls.

5. Adherent pericardium.—The influence of pericardial adhesions in the production of hypertrophy of the left ventricle must be ascribed to the restraint exercised by the attachments to the free movement of the organ. The cardiac hypertrophy is frequently, indeed usually, associated with dilatation from failure, and this is especially pronounced in the case of the right ventricle (see p. 130).

6. Lesions of the right heart and obstructive diseases of the lungs.—The hypertrophy of the left ventricle, which may accompany lesions of the lungs and right heart, has already been accounted for in a preceding section, and need not be further considered here.

Dilatation

Dilatation of the left or right ventricle may be due either to the overfilling or to the incomplete emptying of their cavities. The mechanism of production in each event has already been fully considered under the head of aetiological pathology.

The lesions which lead to dilatation of the left ventricle from overfilling are aortic, and mitral incompetence, and of the right ventricle, pulmonic and tricuspid incompetence.

Dilatation from failure is the result of either the absolute or relative inadequacy of the contractile power of the ventricles in the face of the resistance that they have to overcome. Thus enfeeblement of their muscular walls may render them unequal to the ordinary requirements of the circulation, or the work to be done may be so increased that even the healthy cardiac muscle is unable to cope with it.

An increase in the resistance to the ventricular contraction is more especially operative in the production of dilatation from failure when the additional stress occurs suddenly, as well as with considerable intensity.

The overstrain of the heart, under such circumstances, may be attended with a serious or even fatal amount of dilatation.

The contractile power of the left ventricle may be enfeebled by structural disease of its walls, or by impairment of nutrition, and by toxæmic, nervous, hereditary, and other causes.

The resistance to be overcome, or, in other words, the work to be accomplished by the left ventricle, is increased by any of the conditions that were enumerated under the causes of hypertrophy of this chamber.

Provided the muscular tissue of the heart is sound, and its nutritional vigour unimpaired, the effect of an increase in the resistance to the onward flow of blood, if gradually established, is to produce hypertrophy of the organ.

The subsequent development of dilatation from failure, if it occurs, is explained by the fact that, sooner or later, the nutritional vigour of an hypertrophied heart tends to become impaired by the conditions under which hypertrophy is observed.

On the other hand, if the cardiac muscle be degenerated, or be suffering from any form of malnutrition, an increase in the resistance to the ventricular systole, however gradual its occurrence, is productive of dilatation from failure.

Even the healthy cardiac walls may undergo dilatation from failure, if the rise of the intra-cardiac pressure, or in other words the increase in the resistance to the ventricular systole, be sudden as well as extreme. In such cases, however, in the absence of a rapidly fatal termination, a variable degree of hypertrophy is subsequently developed.

Acute Dilatation

It will be convenient under this head to briefly consider that form of acute dilatation of the heart which is due to mechanical strain.

From a clinical point of view, the effects of mechanical strain of the heart are (1) damage to the orifices and valves of the organ; (2) damage to the myocardium; and (3) functional disturbance of the heart. It is, of course, obvious that these results are more readily produced and more pronounced when the valves and myocardium are already the seat of malnutrition or morbid changes.

Apart from functional disorders of the heart which will be considered later, the effects of acute and chronic valvular and myocardial strain have for the most part been described. In this place it will be necessary only to call attention to acute parietal strain of the heart from over-exertion in those cases, usually young subjects, in which the organ is presumably sound, or at all events has not previously shown any appreciable evidence of insufficiency.

The chief interest and the chief difficulty that attach to acute parietal strain of the heart are the manner in which the stress produced by violent or unwonted exertion tells upon the organ. A detailed consideration of the problem is out of the question, but an attempt will be made to briefly outline the chief factors concerned in the production of cardiac stress and their mode of operation.

The changes in blood pressure that attend muscular exertion exercise, of course, a most potent influence.

Active muscular exercise raises the mean arterial blood pressure at the outset, and subsequently, at a variable interval, lowers or even annuls the rise. The duration of the rise of the mean arterial pressure varies, according to Oliver, with the nature and severity of the exercise, and with the vaso-motor tone of the individual, etc.

The effect of exercise on the venous blood pressure is similar; but the rise of pressure is somewhat more prolonged than on the arterial side of the circulation. Expressed in terms of heart stress, the initial rise of blood pressure adds to the work of the left ventricle in proportion to the extent of the rise; the subsequent fall of pressure diminishes the work of the ventricle. The rise of venous pressure augments the supply of blood to the right side of the heart, and hence by way of the lungs and left auricle to the left ventricle. The total additional work imposed on the left ventricle is therefore the propulsion of an increased charge against increased resistance, while in the case of the right ventricle an increased charge only has to be dealt with.

The influence of the quickening of respiration that attends all forms of muscular exertion is difficult to gauge with anything like precision. Stated generally, inspiration favours the diastolic filling of the right heart, for not only is the blood aspirated from the extra-thoracic veins, but it is also pressed up from the abdomen by the inspiratory descent of the diaphragm. These conditions are

reversed during expiration. Again, so far as the pulmonic circulation is concerned, inspiration, when the breathing is rapid, lessens the supply of blood to the left heart, while expiration increases it. The occurrence of dyspnoea is a source of help to the heart and circulation, inasmuch as each respiration pumps blood from the venous side of the circulation through the lungs into the left heart. The general effect then of the respiratory pressures that attend muscular effort is to still further augment the supply of blood to the right and left sides of the heart, and in this way to increase the work of the ventricles. Finally, in attempting to estimate the effects of prolonged muscular exertion on the heart, it is necessary to take into consideration the influence on the myocardium of the nervous exhaustion that waits upon fatigue, and of the entrance into the circulation of the waste products of muscular metabolism.

It will appear, therefore, that the stress imposed on the heart by muscular exertion consists in the over-filling and consequent over-distension of both the right and left sides of the organ. The left ventricle has also to contend, initially, against increased peripheral resistance. Relief to the left ventricle is afforded by a fall in blood pressure consequent on diminished peripheral resistance, and to the right ventricle by increased expansion of the lungs, which enlarges the bed of the pulmonic circulation, and by the safety-valve action of the tricuspid valve. It now becomes possible to roughly apportion the degree and kind of stress to which each side of the heart is exposed.

Thus at the outset the left ventricle has to cope not only with an increased load, but also with increased resistance to the discharge of its contents. Provided the limits of the reserve power of the heart are not exceeded, the ventricle is able, with more or less difficulty, to propel its charge of blood into the aorta. The subsequent fall of arterial blood pressure enables the ventricle to once more take command of its contents and carry the systole through.

The conditions of stress on the right side of the heart depend for the most part on over-filling, which persists during prolonged muscular effort for considerable periods of time. Distension of the right ventricle from over-filling is a normal accompaniment of all forms of exertion, and is no doubt a cause of the temporary cardiac embarrassment that is usually experienced by most individuals during the early stages of unwonted effort. The fall of pressure in the pulmonic circulation coincident with the expansion of the lungs, and the safety valve action of the tricuspid valve enable the ventricle to rise to the occasion and deal with its increased charge, so that the so-called "Second Wind" is established.

These then are the effects of supportable stress on the heart in the normal or "trained" subject.

If the heart "gives out" the failure may occur on the right or left side or on both sides of the organ.

In cases of sudden and excessive stress the left ventricle may fail

to completely discharge its contents with each systole, so that the chamber undergoes dilatation from incomplete emptying, that is, from failure. The cause of the cardiac failure in these cases depends in all probability on interference with the adjusting vaso-motor mechanisms which make for a reduction in blood pressure. Apart from the considerations which *a priori* lend support to this view, the chief argument in favour of it is the fact that acute dilatation of the heart is not infrequently observed under conditions of stress which seem totally inadequate to explain its occurrence. It has seemed, too, that this event is more especially liable to occur in boys of a nervous temperament.

In searching for an explanation of the apparent anomaly, it may be pointed out that muscular exertion, more particularly among boys, is commonly attended by emotional excitement, which is a well attested and potent source of increase of arterial blood pressure. It is this factor which in the opinion of the writer has not been sufficiently recognised in the causation of acute dilatation of the left side of the heart.

Acute dilatation of the right side of the heart may be contrasted in respect of causation with the similar condition on the left side of the organ, inasmuch as it is the outcome of prolonged muscular effort. Allbutt, who originally called attention to the effects of mechanical strain of the heart, believes that, to all intents and purposes, dilatation of the right side of the heart is a normal incident of prolonged exertions.

The occurrence of acute dilatation of the right heart under these circumstances is explicable on the grounds that the over-charging and consequent distension of the ventricle is so great that the chamber is unable to completely discharge its contents with each systole, and consequently undergoes dilatation from incomplete emptying.

This event is more likely to happen when, owing to an insufficient increase in the capacity of the lungs, the resistance in the pulmonic circulation does not fall enough to bring the work of the ventricle within the limits of its reserve power.

The safety valve action at the tricuspid opening must in the nature of the conditions that obtain quickly become exhausted, so far as any relief to the right ventricle is concerned. No doubt, too, in cases of prolonged exertion the effects of the waste products of muscular metabolism and of nervous exhaustion contribute their quota to the undoing of the ventricle.

It is doubtful whether a heart that has undergone acute dilatation ever completely recovers its tone and functional vigour. In most instances, at all events, some disability is left behind which renders the organ unable to cope with stress which falls well within the physiological powers of a normal heart. The explanation may be that interference with the functional capacity of an organ is always productive of a greater or less degree of interstitial fibrosis which must act as a drag on functional efficiency.

RIGHT VENTRICLE

Hypertrophy

1. **Valvular disease on the right side of the heart.**—The valvular diseases which give rise to hypertrophy of the right ventricle are pulmonary stenosis and pulmonary incompetence, and tricuspid insufficiency, as well as any combinations of these lesions. The mode of production of the ventricular hypertrophy resembles in all respects that described for the corresponding affections on the left side of the heart. Hypertrophy of the right ventricle, except in cases of pulmonary stenosis, is usually attended with a considerable degree of dilatation.

2. **Valvular disease on the left side of the heart.**—The rise of pressure in the pulmonary circulation induced by mitral and eventually by aortic lesions, necessitates an increase in the work of the right ventricle, which consequently undergoes hypertrophy.

Mitral disease, predominantly mitral stenosis, is more efficacious in this respect than aortic.

The hypertrophy of the ventricle is seldom found in a pure state, that is to say, it is usually combined with dilatation from failure.

3. **Diseases of the lungs.**—The pulmonary affections which interfere with the passage of blood from the right to the left side of the heart, and thus increase the work of the right ventricle and give rise to its hypertrophy, are chiefly bronchitis, and emphysema, consolidation, collapse, and fibrosis of the lungs.

The ventricular hypertrophy is almost always associated with more or less dilatation from failure, which in many cases is the primary lesion.

4. **Adherent pericardium.**—The effect of pericardial adhesions in the production of cardiac enlargement is more marked in the case of the right ventricle than of the left. The reasons for this are that the comparatively thin walls of the right ventricle suffer more from the primary inflammation and are less capable of resistance than the thicker-walled left ventricle, and further, that the fibrous attachments act over a relatively large superficial area in the case of the former chamber as compared with the latter.

Dilatation from failure is always associated with the ventricular hypertrophy, and is not infrequently the predominant condition.

Dilatation

The right ventricle is more liable to undergo dilatation from failure than the left. This is accounted for by the proportionately diminished power of resistance to strain possessed by the thinner walls of the right ventricle, and also by the fact that the nutritive

processes associated with the conditions under which the chamber is exposed to stress, are seldom favourable for the production or maintenance of an adequate degree of hypertrophy.

The explanation given of the mode of production of dilatation of the left ventricle applies, *mutatis mutandis*, with equal force to the right.

All the causes of hypertrophy of the right ventricle lead, sooner or later, to dilatation of the chamber from failure. Dilatation of the right ventricle from overstrain consequent on severe and prolonged muscular effort is by no means uncommon, and in extreme instances a rapidly fatal termination is sometimes observed on account of the supervention of asystole.

LEFT AURICLE

Hypertrophy

The most common cause of hypertrophy of this chamber is mitral stenosis, in which disease the thickness of the auricular walls has been known to exceed one quarter of an inch. The hypertrophy of the auricle is usually accompanied by a variable degree of dilatation from failure.

Hypertrophy in association with dilatation is observed, also, in cases of mitral regurgitation.

Dilatation

Dilatation of the left auricle accompanies all forms of mitral disease, whether obstructive or regurgitant.

RIGHT AURICLE

Hypertrophy

Hypertrophy of the right auricle, with dilatation, is seen in cases of tricuspid stenosis and of primary incompetence of the valve.

Dilatation

Insufficiency of the tricuspid valve, in whatever manner produced, or narrowing of the opening, leads to dilatation of the right auricle.

SYMPTOMS

Hypertrophy

So long as hypertrophy is efficient, that is, so long as there are no indications of embarrassment of the circulation, or of respiratory distress or disturbance, during rest or moderate exertion, symptoms

are few and unimportant. A feeling of fulness, uneasiness, constriction, or pressure, amounting in some instances to actual pain, may be experienced in the præcordial region.

Palpitation of the heart is easily excited, and may be accompanied by rapid or irregular action of the organ. A dry, irritating cough, with or without a sense of respiratory oppression, is sometimes a source of much discomfort.

Throbbing sensations in the head and neck, noises in the ears, vertigo, or *muscæ volitantes* may be complained of, especially after unusual muscular or mental effort. Sleeplessness is sometimes a troublesome symptom.

Epistaxis, hæmoptysis, hæmatemesis or *melæna*, and cerebral hæmorrhage may occur as the result of arterial rupture, consequent on the strain to which these vessels are exposed in cases of cardiac hypertrophy.

Other symptoms which may be observed are due less to the hypertrophy of the heart than to the causes that have given rise to it.

The earliest indication of commencing failure of compensation is the occurrence of dyspnœa on slight provocation, but it must be borne in mind that the presence of this symptom, without other evidence, is no proof of the existence of heart disease.

Dilatation

It will be advantageous to trace the symptoms which attend dilatation from failure in the order of their usual occurrence, from the left to the right side of the heart, and from the time when failure of compensation begins.

During the early stages of the process the patient complains of shortness of breath on slight exertion, accompanied by præcordial uneasiness or pain, palpitation and irregular action of the heart. The imperfect supply of blood to the brain gives rise to lassitude, headache, drowsiness, or sleeplessness, and attacks of giddiness or faintness.

Digestive derangements, manifested by a furred tongue, loss of appetite, nausea, pain after food, flatulence, and irregular action of the bowels are among the earliest indications of commencing failure of compensation. Flatulent distension of the stomach may be a source of great discomfort, or even of danger, by reason of the interference with the free action of the heart. The development of mitral incompetence leads to a general aggravation of the symptoms. The shortness of breath becomes more pronounced and more easily excited, and is now attended by cough, hæmoptysis, and the signs of engorgement of the pulmonary circulation.

The appearance of the patient at this stage of the disease is often highly suggestive of imperfect aëration of the blood. Thus the cheeks, lips, nose, and ears present a dusky bluish red or ashy hue; the hands and feet are cold and livid. Nutrition is seriously inter-

fered with, and the heart suffers in common with the other organs of the body, to the detriment of the compensatory changes and of the patient generally.

Wasting may be observed, more especially in the case of children.

Failure of the right heart is followed by a further series of events, which culminate in portal and systemic venous congestion and dropsy.

Shortness of breath, which may amount to orthopnœa, is now a constant and distressing symptom.

The congestion of the portal circulation leads to further functional derangement of the liver, stomach, and intestines, with the production of jaundice, persistent dyspepsia, and constipation or diarrhœa.

The urine is high-coloured, diminished in quantity, and shows on standing a copious deposit of urates. It usually contains albumen and occasionally blood.

Dropsy commences in the feet and gradually spreads upwards. The loose cellular tissues of the scrotum and lumbar region are often enormously distended with fluid. Dropsical effusion may also take place into the serous cavities of the pleuræ, pericardium, and peritoneum, events which add greatly to the cardiac embarrassment.

Hæmorrhages sometimes occur into the subcutaneous tissues, and the œdematous skin may be the seat of erysipelatous inflammation, or even of gangrene.

Epistaxis, hæmatemesis, or melæna, menorrhagia, etc., are observed as the result of venous or capillary rupture.

The signs and symptoms of the embolic plugging of one of the arteries of the spleen, kidneys, brain, or lungs may appear at any stage of the disease.

Death is usually due to a process of slow asphyxia dependent on the gradual enfeeblement of the cardiac walls. The fatal termination is sometimes precipitated by an acute pulmonary or pericardial complication, or by some accidental circumstance, such as embolism of a cerebral artery, etc.

Excluding valvular affections, sudden death is rarely observed, except as the occasional result of fatty disease of the myocardium, or extensive pericardial adhesions.

In cases of primary tricuspid regurgitation, and of disease of the right ventricle, affecting that chamber exclusively or predominantly, Sir William Broadbent has observed (Lumleian Lectures, 1891) that the symptoms have been due rather to an insufficient supply of blood to the left ventricle, giving rise to a tendency to syncope, than to damming back of blood in the veins.

The symptoms of acute dilatation of the heart are sudden acute pain, or a feeling of great oppression or of something giving way in the chest, accompanied by pallor of the face, collapse, sweating, rapid shallow breathing, or a peculiar gasping respiration, restlessness, delirium, together with coldness and lividity of the extremities.

PHYSICAL SIGNS

HYPERTROPHY OF THE LEFT VENTRICLE

There is nothing particularly characteristic about the appearance of patients suffering from hypertrophy of the heart, apart from the conditions that have given rise to it.

The pale, puffy face of Bright's disease, or the greyish-white complexion associated with aortic lesions, could not fail to strike the observer in a case of cardiac hypertrophy, of which either of these affections was the cause.

Pulse

The character of the pulse varies with the condition which has given rise to the hypertrophy.

In high arterial tension the artery, which may be small or large, is full between the beats, and can be rolled beneath the finger; the pulse wave rises and falls slowly, and is not easily obliterated by pressure. The coats of the vessel may be normal, but, as a rule, they are more or less thickened and distorted.

Extensive fibroid degeneration, with loss of elasticity of the arterial walls, will modify the characters of the pulse; for although the vessel still remains full between the beats, the wave now rises and falls quickly.

In aortic incompetence the artery is large, empty between the beats, and the pulse wave rises and falls abruptly, giving the peculiar sensation of collapse so characteristic of this affection.

In aortic stenosis the artery is small, and can be felt between the beats; the pulse wave rises and falls slowly, and is not easily obliterated by pressure.

The pulse of mitral regurgitation is irregular, both in force and frequency, while the artery is of variable size, and, as a rule, cannot be felt between the beats.

Heart

Inspection.—There may be prominence or even bulging of the præcordial area. This is most commonly observed in the case of children, whose chest wall is softer and more capable of yielding than that of adults.

The apex beat, if visible, is seen to be displaced downwards and somewhat outwards, and may be situated in the sixth or seventh intercostal space, or even lower. Systolic retraction of the intercostal spaces immediately above, and internal to the apex beat, is occasionally, though rarely, observed.

The impulse of the ventricle is a deliberate circumscribed push or heave, frequently strong enough to visibly raise the chest wall.

Pulsation may be observed in the carotid arteries of the neck, more especially in the case of aortic regurgitation.

Palpation.—The position of the apex beat can now be exactly determined by the finger.

The impulse of the ventricle is felt as a prolonged, powerful, localized thrust.

Pulsation may be detected immediately to the right of the sternum, in the first and second interspaces, when dilatation of the aorta is associated with hypertrophy of the left ventricle. The character of the ventricular impulse may be masked by overlapping lung in cases of emphysema.

Percussion.—The area of cardiac dulness is increased downwards and to the left.

Auscultation.—At the apex the first sound is duller and more prolonged than normal, probably because the muscular element of the first sound now predominates over the valvular. In the absence of aortic valvular disease, the second sound at the apex is usually more or less accentuated.

Persistent reduplication of the first sound of the heart is indicative of commencing failure of the left ventricle.

At the base over the aortic cartilage, the first sound is indistinct, or altogether inaudible; the second sound is heard louder than normally. If the root of the aorta is dilated the second sound is not only accentuated, but has in addition a low-pitched ringing quality.

The association of a weak, indistinct aortic second sound with high arterial tension and a vigorous hypertrophied ventricle strongly suggests the coexistence of aortic valvular disease.

The adventitious auscultatory phenomena which attend hypertrophy and dilatation of the heart in cases of valvular disease have been described elsewhere, and will not be reconsidered, otherwise than incidentally, in this section.

DILATATION OF THE LEFT VENTRICLE

Physiognomy

The subjects of cardiac dilatation present, sooner or later, the appearances described under the head of mitral regurgitation. In advanced cases the patient exhibits varying degrees of cyanosis and dropsy.

An anxious, distressed expression, watery eyes, with puffy lower lids, pale or bluish lips, cheeks and nose studded with congested capillaries, cold and livid extremities, often more or less œdematous, and an attitude suggestive of difficulty in breathing, constitute, severally or collectively, the appearances observed from time to time in the different stages of dilatation of the heart. It is by attention to details of this kind that the diagnosis of the patient's ailment can often be made, without the help afforded by further examination.

Pulse

The characters of the pulse in dilatation of the left ventricle vary not only with the cause of the condition, but also with the degree of its development.

With these qualifications, it may be stated generally that the pulse of dilatation is usually rapid and irregular, both in force and frequency; and if not, that it tends to become so on slight provocation. The artery, which may be large or small, cannot be felt between the beats, except in cases of antecedent high arterial tension, and in this event the vessel, though palpable between the beats, is easily compressible. The pulse wave is sudden, short, and badly sustained, and is easily obliterated by gentle pressure with the finger.

Heart

Inspection.—There may be bulging or retraction of the intercostal spaces over some portion of the præcordial area. The apex beat, when visible, is seen to be displaced outwards and downwards. The impulse of the left ventricle is usually indefinite and diffused over a larger area than in health, or it may be altogether imperceptible. Pulsation in the epigastrium and over the right ventricle is frequently observed.

Palpation.—The localization of the apex beat should be attempted by means of the finger, but it is often impossible to exactly define its position owing to the weakness of the ventricular contraction. The impulse of the ventricle is either uncertain, feeble, and tapping in character, or it may be felt as a vague diffuse vibration spread over a considerable area. In some instances the ventricular impulse is not perceptible, even on careful palpation, and provided this is not due to overlapping of the heart by lung, it is an indication of great enfeeblement of the cardiac walls.

An enlarged right ventricle may usurp the place of the left, and in this way it sometimes happens that the site of maximum intensity of the cardiac impulse to the left of the sternum corresponds with some portion of the wall of the former chamber instead of the latter.

Forcible epigastric pulsation would point to hypertrophy of the right ventricle, whereas a feeble vibratory impulse in this situation would indicate dilatation of the right side of the heart.

Percussion.—The area of cardiac dulness is increased, chiefly in the direction of the left axilla, but also downwards. The left limit of percussion dulness may extend outwards as far as the mid-axillary line. Downwards it sometimes reaches as low as the seventh or eighth interspace. Extension of the right limit of cardiac dulness, if observed, is due to concomitant enlargement of the right side of the heart.

Auscultation.—The first sound at the apex is short, sharp, and louder than normal, which is due, no doubt, to the absence, in a greater or less degree, of its muscular element. If the dilatation of the ventricle has given rise to mitral incompetence, the first sound may be masked by a systolic murmur. The second sound at the apex is usually weak. At the base over the aortic cartilage both sounds of the heart will be audible, and it is sometimes difficult to distinguish between them, since the character of the first sound in dilatation closely resembles that of the second. The second sound in this situation tends to become weaker with the increasing enfeeblement of the muscular power of the ventricle, hence a comparison of the relative intensity of the two sounds may become a point of diagnostic and prognostic value. The pulmonary second sound is accentuated, provided the right ventricle has not undergone dilatation from failure.

Important information with regard to the vigour of the ventricular walls may be obtained by a careful observation of the interval between the first and second sounds of the heart. Thus the interval may be prolonged until the sounds become equidistant, or it may be shortened, so that the second sound follows the first almost immediately.

Prolongation of the interval, or spacing of the sounds, as it is called, signifies that the systole of the ventricle is lengthened in consequence of the difficulty experienced by the chamber in the discharge of its contents, and demonstrates that the ventricle, though hampered by the increased demands upon it, is attempting successfully to complete its contraction.

Approximation of the sounds, under the circumstances, now being considered, means that the contractile energy of the ventricular walls becomes exhausted before the completion of the systole. So soon as the pressure in the aorta exceeds that in the ventricle, an event determined by the relative degree of inadequacy of the cardiac walls, the semilunar valves close, and the second sound occurs.

It will be obvious that approximation of the sounds is of much graver significance than prolongation of the interval between them.

The phenomena just described are most commonly observed in cases of continued high arterial tension, the result of acute or chronic Bright's disease.

HYPERTROPHY OF THE RIGHT VENTRICLE

Physiognomy

The aspect presented by the patient is that of the condition which has given rise to the hypertrophy of the right ventricle. Since, however, all the causes of right-sided hypertrophy of the heart tend to interfere with the pulmonary circulation, the appearance of the patient is frequently suggestive of mal-aëration of the blood.

Pulse

Hypertrophy of the right ventricle exercises no direct influence upon the radial pulse. The modifications in the character of the pulse that may be observed are due not to the hypertrophy of the ventricle, but to the accompanying obstruction to the blood current in the pulmonary circulation.

One effect of this is to diminish the supply of blood to the left ventricle, and hence to the aorta and peripheral vessels; another is to produce general contraction of the systemic arterioles, in consequence of the stimulation of the vaso-motor centre by the imperfectly oxygenated blood.

The radial artery, therefore, in cases of hypertrophy of the right ventricle, is small, and though palpable between the beats is easily compressible. The pulse may or may not be regular in force and frequency, while the wave is small and frequently short.

Heart

Inspection.—The epigastric region is sometimes prominent, and there may be bulging of the ensiform cartilage and of the lower left costal cartilages at their junction with the sternum.

Pulsation is often observed in the epigastrium, or between the lower part of the left sternal edge and the apex beat.

The jugular veins may be distended, and if the tricuspid valve is incompetent may show pulsation. The apex beat is seen to be displaced to the left.

Palpation.—The hand placed in the epigastrium or over the right ventricle experiences the sensation of a forcible push or thrust.

Hepatic pulsation may be due to the impulse of the right ventricle transmitted through the diaphragm, or to regurgitation of blood into the portal veins, consequent on tricuspid incompetence.

Percussion.—The area of cardiac dulness is increased chiefly in the transverse direction, but also to some extent downwards. To the right it may extend an inch or more beyond the right sternal edge.

Auscultation.—The first sound over the right ventricle is duller and louder than in health, so that it approaches in character the normal left ventricle first sound. If the tricuspid valve is incompetent, the first sound is accompanied, and more or less masked, by a systolic murmur.

At the base, over the pulmonary cartilage, the second sound is accentuated, so long as the tricuspid valve remains competent, and in the absence of valvular disease at the pulmonic orifice. The aortic second sound is usually weak.

Reduplication of the first or second sound of the heart is not infrequently observed in cases of hypertrophy of the right ventricle. The interval between the two sounds should in all cases be carefully noted, for the reasons already given.

DILATATION OF THE RIGHT VENTRICLE

Physiognomy

The patient usually presents in a greater or less degree the signs of cyanosis and dropsy.

Pulse

The pulse, though not directly affected by dilatation of the right ventricle, is greatly influenced by the associated lesions in the lungs or on the left side of the heart.

As a rule, therefore, the pulse is rapid, and irregular both in force and frequency. The artery is usually small, and when palpable between the beats is easily compressible.

The pulse wave is small, short, and weak.

Heart

Inspection.—There may be bulging of the lower part of the sternum, and of the immediately adjacent costal cartilages.

The epigastrium is usually prominent and not infrequently shows pulsation.

The impulse of the right ventricle is diffused over the portion of the præcordial area between the apex beat and left sternal edge as high as the second interspace. Pulsation to the right of the sternum in the second and third interspaces may be due to an enlarged right auricle.

The veins in the neck appear distended, and there may be jugular and hepatic pulsation.

Palpation.—The impulse of the right ventricle felt over the lower portion of the sternum and in the epigastrium is feeble, tapping, and diffused, or it may be quite imperceptible. The jugular veins may fill from below, when emptied by pressure with the finger from below upwards, in consequence of regurgitation of blood through the tricuspid opening. A similar cause can give rise to pulsation of the liver, which is sometimes observed.

Percussion.—The area of cardiac dulness is increased chiefly in the lateral direction, and may extend an inch or more to the right of the sternum.

Auscultation.—The first sound over the right ventricle is short and sharp, and it may be accompanied by a systolic murmur, which

is most distinctly heard over the ensiform cartilage and lower portion of the sternum.

The pulmonary second sound loses its accentuation with the occurrence of tricuspid incompetence. The aortic second sound is usually weak.

HYPERTROPHY AND DILATATION OF THE LEFT AURICLE

Since hypertrophy of the left auricle is almost invariably found in association with dilatation of its cavity, it will be convenient and sufficiently accurate to consider the physical signs of enlargement of the chamber, irrespective of its exact mode of causation.

A similar plan will be followed in the case of the right auricle.

It may be pointed out, however, that as a rule dilatation preponderates over hypertrophy in all forms of auricular enlargement. It is stated that pulsation in the third left interspace, due to the systole of an enlarged left auricle, may sometimes be observed in the case of children and of persons with thin chest walls. Dulness in the second and third left intercostal spaces close to the sternum has been ascribed to enlargement of the left auricle, but this is not admitted by all observers.

HYPERTROPHY AND DILATATION OF THE RIGHT AURICLE

Enlargement of the right auricle gives rise to dulness in the second, third, and fourth intercostal spaces, extending for an inch or more to the right of the sternal edge. Pulsation may be caused in this situation by the systole of an enlarged auricle.

In some cases of tricuspid obstruction the hypertrophy of the auricle may be sufficient to give rise to a backflow of blood into the veins during the systole of the chamber.

In such an event the venous pulsation would, of course, be presystolic as regards its relation to the cardiac cycle.

DIAGNOSIS

The first step in the diagnosis is the exclusion of the conditions which may simulate or mask enlargement of the heart. If, in this way, the diagnosis of cardiac enlargement is established, three points remain to be considered, viz. (1) whether the increase in the size of the heart is due to hypertrophy or to dilatation, (2) whether the enlargement of the organ is partial or general, and (3) the cause of the increase in size of the heart.

The conditions which may simulate or mask enlargement of the heart will now be considered.

Bulging of the chest wall involving the præcordium may be due to congenital malformation, rickets, mediastinal tumours, left pleural effusion, etc.

Attention to shape, position, and outline of the prominent areas arising from these causes is usually sufficient to distinguish them from the præcordial bulging associated with diseases of the heart and pericardium.

For similar reasons the recession of the præcordium due to congenital malformations, retraction of the left lung, and long-continued pressure in this situation, as in the case of shoemakers, joiners, etc., will not be confounded with the effects of pericardial adhesions.

In a case of doubt, the history of the patient would be of great assistance in clearing up the diagnosis.

It has already been mentioned that the dulness due to cardiac enlargement may be masked by the overlapping of the heart by lung. The recognition of the existence of pulmonary emphysema would suffice to exclude this source of error.

Enlargement of the heart may be simulated by exposure of the organ, consequent on retraction of the anterior margins of the lungs, by consolidation of the anterior portions of either lung, by local pleural effusion, by aneurism of the ascending portion of the aorta or by mediastinal tumours above the base of the heart, by forward displacement of the organ, and by pericardial effusion.

Except in the case of pericardial effusion, the differential diagnosis is not usually a matter of very great difficulty, and depends on the history of the patient and on a careful estimation of the extent and outline of the areas of dulness, and also on the associated symptoms and physical signs.

Pericardial effusion may be distinguished by the shape of the outline of dulness and its relation to the apex beat, by the position and character of the cardiac impulse, by the presence of friction sounds, and by the history and associated conditions (see p. 125).

Displacement of the heart and apex beat to the left due to retraction of the left lung, effusion of air or fluid into the right pleural cavity, elevation of the diaphragm, and aneurism of the ascending aorta may be confounded with cardiac enlargement.

Under these circumstances, however, the area of cardiac dulness, though dislocated, is not increased, and this fact, taken in conjunction with the presence of a cause for displacement of the heart, and the absence of any discoverable cause of enlargement, should go a long way to remove any difficulty that may be experienced in forming a correct diagnosis.

The Differential Diagnosis of Hypertrophy and Dilatation

Although hypertrophy and dilatation are so frequently found in combination, it is convenient for clinical purposes to consider a heart hypertrophied or dilated according as one or other condition predominates.

In attempting to decide which of these conditions preponderates in the production of cardiac enlargement, reliance should be placed chiefly on the evidence afforded by the shape of the præcordial dulness, and by the character of the impulse and of the sounds, together with the absence or presence of the signs of disturbance of the circulation.

It may be stated generally that hypertrophy or dilatation predominates according as enlargement of the heart exists without or with the signs of circulatory embarrassment.

The Distribution of Hypertrophy and Dilatation in Enlargement of the Heart

All or any of the chambers of the heart may be affected by hypertrophy or dilatation.

The diagnosis of the general or partial involvement of the organ rests on the causes of the cardiac enlargement, considered in conjunction with their known effects, and on the symptoms and physical signs which are associated with hypertrophy and dilatation of the different chambers of the heart.

The Cause of the Cardiac Enlargement

The importance that attaches to the elucidation of the cause of enlargement of the heart cannot be too strongly insisted upon, since it is on this point that the prognosis and treatment largely turn.

In those instances in which cardiac enlargement is due to disease of the heart, lungs, or kidneys, the cause is as a rule easily recognized.

It is in the diagnosis of the source of dilatation from failure that difficulty most commonly arises. Here not only must primary dilatation of the heart be distinguished from dilatation secondary to valvular or pericardial disease, etc., but the precise cause of the failure of the muscular walls, in any event, must if possible be determined.

The difficulties that may attend this part of the diagnosis can be most conveniently illustrated by the study of a concrete example.

Displacement of the apex beat to the left, with a corresponding extension of the left limit of cardiac dulness and an apical systolic murmur, are the physical signs associated with regurgitation through the mitral opening, due to valvular disease or to muscular incompetence, consequent on primary dilatation of the left ventricle.

The differential diagnosis is a matter of great importance on account of its bearing on the prognosis, which is very different in the two conditions, since the one is permanent and irremediable, whereas the other is often temporary and curable.

The character and extent of propagation of the murmur, with its relation to the first sound, the character of the pulse, the causal indications, and the effects of treatment are the points which afford the most reliable means of distinction.

Thus a loud, well-conducted murmur, partially or wholly obscuring the first sound; a small, weak, irregular pulse; a definite history of rheumatic fever; and an increase in the intensity of the murmur under treatment, would point to valvular incompetence.

On the other hand, a clear, short, and sharp first sound, followed and not obscured by the murmur, which is not well conducted towards the left axilla; a pulse of variable character, but possibly exhibiting increased tension; the presence of anæmia, or other cause of malnutrition; and the disappearance of the murmur under treatment, would indicate muscular incompetence.

In either event the exact cause of the dilatation from failure should be diligently sought for.

It may depend on the operation or co-operation of many forces, chief among which are mental or bodily over-exertion, excitement, anxiety, exposure, chill, dyspepsia, anæmia, or malnutrition from other causes, and pulmonary or other complications. Indiscretions in eating and drinking, sexual excess, neglect of the action of the bowels, the abuse of alcohol, tea, tobacco, contribute towards the same result.

PROGNOSIS

The attempt to forecast the course and issue of affections of the myocardium is no whit less important than in the case of valvular disease of the heart.

The problem in myocardial lesions is, however, rendered more complicated by the fact that no such correlation exists between the extent of the disease and its effects on the heart and circulation as obtains in valvular affections.

In the absence of this important source of knowledge, the prognosis in myocardial disease turns on collateral considerations of cause and effect, which, though productive of valuable evidence, lack the exactitude and precision essential to the formation of a reliable forecast of the probable course of events.

However, in spite of these difficulties, it is frequently possible, by means of a careful and complete survey of all the phenomena associated with lesions of the cardiac walls, to arrive at a fairly accurate estimate of their course and duration.

The subject will be considered under the heads of hypertrophy and dilatation respectively.

Hypertrophy

Provided that compensation is efficient, and also that the conditions, both local and general, are favourable for the maintenance of hypertrophy, the prognosis is favourable.

In estimating the probable duration of compensation, the age, sex, occupation, and condition in life of the patient must be taken into account, as well as the nature and tendency of the morbid change which has given rise to the hypertrophy.

It sometimes, though rarely, happens that the cause of the hypertrophy is partially or wholly removable, and, other things being equal, the outlook is propitious in proportion to the degree of this possibility.

As a rule, however, the cause is abiding, and furthermore, it may be either stationary or progressive.

The prognosis, speaking generally, is more favourable in the event of the first alternative than in that of the second.

It must be borne in mind that, in cases of cardiac hypertrophy due to high arterial tension, the rupture of a vessel in the brain or elsewhere may upset an otherwise favourable prognosis.

Dilatation from Failure

In attempting to forecast the issue of dilatation of the heart from failure, it is necessary to carefully weigh and compare the indications derived from the following sources of information :—

1. The general state of the patient.
2. The condition of the cardiac muscle as shown by the symptoms and physical signs.
3. The cause of the failure of the cardiac walls.
4. The history, habits, etc., of the patient.
5. The presence or absence of complications.
6. The effects of treatment.

Each of these factors in the prognosis will now be considered in detail.

1. **The general state of the patient.**—The severity of the symptoms corresponds with the degree of disturbance of the circulation, and hence with the extent to which the propulsive power of the heart is impaired.

Indications of impending danger may appear on the venous or on the arterial side of the circulation, and among the signs and symptoms of serious import may be mentioned extensive water-logging of the tissues, severe and protracted vomiting, continued sleeplessness, great bodily weakness, and attacks of faintness or extreme breathlessness.

2. **The condition of the cardiac muscle as shown by the symptoms and physical signs.**—The estimate so far made of the degree of efficiency of the cardiac muscle must be supplemented by an examination of the pulse and heart. Extreme irregularity of the pulse, or a great increase in frequency, if long continued, is of bad omen.

Disappearance of previously existing high arterial tension, not due to treatment, implies failure of the muscular power of the heart, and is therefore an unfavourable sign.

The degree of cardiac vigour is gauged more accurately by the strength of the impulse and by the extent of the percussion dulness, together with the character and time relations of the sounds of the heart.

A feeble, hesitating impulse, great increase in the lateral extent of dulness, and approximation or weakness of the sounds, are indicative of serious enfeeblement of the cardiac walls, and warrant therefore a serious prognosis.

On the other hand, a fair strength of impulse, with some accentuation or spacing of the sounds, implies a certain degree of cardiac vigour, and consequently renders the outlook more hopeful.

3. **The cause of the failure of the cardiac walls.**—If the cause of failure can be discovered and removed the prognosis will be favourable. Thus the effects of over-exertion, anæmia, or malnutrition, dyspepsia, etc., may be remedied by suitable treatment, and in this way the muscular power of the heart may be restored and the normal balance of the circulation re-established.

On the other hand, if the enfeeblement of the cardiac walls proceeds from irremovable causes, as, for instance, acute intercurrent disease or radical weakness of the heart, the outlook will be most discouraging.

4. **The history and habits of the patient.**—The age, history, and habits of the patient exercise an important influence on the course of the disease. Youth, a good family history, a regular mode of life, and abstemious habits will tell in favour of recovery. On the other hand, a constitution lacking hereditary vigour, and undermined by exposure, the abuse of alcohol, etc., would offer but a small margin of recuperative power.

5. **The presence or absence of complications.**—Concurrent disease of the kidneys, lungs, or liver would detract greatly from the prospects of recovery, not only by reason of the increased strain which is thrown on the heart, but also on account of the general interference with nutrition and metabolism, due to functional derangement of these organs.

The absence of complications is of favourable import.

6. **The effects of treatment.**—The manner in which the heart responds to treatment affords an important indication of the con-

dition of the cardiac muscle. A well-marked and rapidly produced beneficial effect implies that the walls of the organ are still sound, whereas the absence of any response to treatment is significant of serious deterioration of the muscular tissue of the heart.

TREATMENT

Hypertrophy

In a previous section it was pointed out that hypertrophy of the heart, although it may be efficient or inefficient, never exceeds the requirements of the occasion which has given rise to it. *Per se*, therefore, hypertrophy does not call for treatment, but rather suggests it. Hypertrophy is the natural method of combating disease, and, as such, should be sedulously encouraged and maintained.

The treatment indicated by hypertrophy consists, then, in the promotion and preservation of the integrity of the cardiac muscle fibre and in the removal or modification, so far as possible, of the causes which have given rise to the increase in the size of the heart.

The conditions which conduce to the furtherance of the first object may be summed up under the heads of hygienic, dietetic, and medicinal treatment, and will now be briefly considered.

Hygienic treatment.—The mode of life and habits of the patient should be regulated with the view of promoting the general health and of avoiding, to the greatest possible extent, the causes of cardiac strain and excitement.

The action of the skin, kidneys, and bowels must be carefully attended to and occasionally assisted by suitable therapeutic means.

Chill should be guarded against by the use of woollen under-clothing, and the incidence of acute disease, so far as possible, prevented.

Acute intercurrent affections, more especially rheumatism and pulmonary disorders, should be vigorously treated, the heart meanwhile being carefully watched for any indications of failure, which, when observed, should be met by the exhibition of stimulants and cardiac tonics.

The patient should be surrounded by wholesome nervous influences and removed so far as practicable from all sources of mental worry and excitement.

While a moderate amount of exercise is beneficial, over-exertion in any form must be rigidly eschewed.

It is by the intelligent application of hygienic measures of this kind that tranquillity of the circulation is promoted and the strain on the heart reduced to the lowest possible limits.

Dietetic treatment.—The diet should be nutritious, non-stimulating, and easily digestible, and ought to contain an ample proportion

of nitrogenous food. Alcohol may be taken sparingly, and at meal times only. Over-eating and drinking must be scrupulously avoided.

Errors of digestion should be corrected without delay, for not only does dyspepsia do harm by interfering with nutrition, but by giving rise to flatulence it directly embarrasses the heart's action.

Medicinal treatment.—One of the chief desiderata in cases of cardiac hypertrophy is an adequate supply of healthy blood to the myocardium. If this object is not achieved by the measures already indicated, it must be promoted by medicinal means. The drugs most useful for the purpose are iron and arsenic, which may be administered either separately or in combination with one another, or with some cardiac tonic such as digitalis or strophanthus. During the use of these remedies the digestive organs should be attended to and the bowels kept open by mild saline and aloetic aperients.

Excited action of the heart may be controlled by the application of cold or a belladonna plaster to the præcordium, and the internal administration of dilute hydrocyanic acid, belladonna, bromide of ammonium, or small doses of opium. The use of cardiac depressants for this purpose may be attended with danger, and should be avoided.

The treatment of the cause.—In the majority of instances hypertrophy of the heart depends upon causes which are organic, permanent, and irremovable. Under such circumstances the treatment already indicated will do much to diminish the strain upon the heart and to encourage and maintain the existing hypertrophy of the organ. Beyond this little or nothing can be effected by therapeutic means.

In those cases, however, in which hypertrophy of the heart is due to prolonged muscular effort or high arterial tension a much greater opportunity is afforded for treatment.

In the first event, the removal of the cause and the substitution of complete rest will usually suffice for the disappearance of the hypertrophy.

Again, high arterial tension may be removed, diminished, or controlled with a corresponding degree of benefit to the condition of the heart.

Since high tension in the arterial system is due for the most part to the presence in the blood of imperfectly oxidised nitrogenous waste products, the aim of treatment is to remove these impurities and prevent their re-formation.

To this end fresh air and regular exercise in one form or another are essential.

The diet must be regulated with the object of restricting the amount of nitrogenous food, which, in the shape of the flesh of beast or bird, should not be taken more often than once a day. Strict moderation must also be observed with respect to the use of alcoholic beverages.

In those instances in which plethora is a factor in the production of high arterial tension, it may become necessary to limit the amount of fluid taken at meals.

The medicinal means by which the products of imperfect nitrogenous metabolism are got rid of, and the resistance to the onward flow of blood through the small arteries and capillaries is diminished, consist in the administration of purgatives and other eliminants.

The most useful drug for this purpose is mercury, preferably in the form of calomel, blue pill, or grey powder, which may be given in doses of two or three grains, once or twice a week, in combination with a course of mild saline aperients, such as the phosphate or sulphate of soda.

The salts of sodium, lithium, and potassium, more particularly the latter, have valuable eliminative properties, and may be prescribed, from time to time, in conjunction with the above-mentioned remedies.

The reduction of peripheral tension may also be effected by the direct abstraction of blood, or by the administration of nitroglycerine, erythrol tetranitrate, and the nitrites, which act by producing relaxation of the muscular coats of the arterioles.

Dilatation from Failure

The treatment of dilatation of the heart from failure may be approached in one of two ways, *i.e.* from the side of the cause or of the effects of the morbid process.

Either procedure has its advantages, but, speaking generally, the first method is better suited to the slighter degrees of dilatation, while the second is more adapted to the advanced stages of the affection. The reason for this is, that in the one case the treatment of the cause is usually sufficient to undo and remove the effects of the lesion, whereas in the other it is not. Moreover, the urgency of the symptoms in advanced dilatation do not admit of the delay which the discovery and treatment of the cause often entail.

Nevertheless, in many instances it is possible to treat both the cause and effects of dilatation simultaneously, and where this is feasible it should be done. If, however, the cause of the morbid process cannot be discovered, or is irremediable, treatment must of necessity be directed towards the effects.

The treatment of dilatation of the heart from failure will therefore be considered under the heads of "Cause" and "Effects," and this division of the subject is not only convenient for descriptive purposes, but also offers many practical advantages, not the least being that it forms the basis of rational therapeutics.

Treatment of the cause.—Dilatation of the heart from failure may be due to increased intra-cardiac pressure, consequent on organic valvular disease or high arterial tension, or it may depend

on impairment of the muscular power of the heart, the result of overstrain, malnutrition, or of structural changes in its walls.

The cause, in the case of organic valvular disease, is permanent and irremediable, and requires, therefore, no further consideration.

The important influence exerted by high arterial tension in the production of dilatation of the heart has already been insisted on, and the treatment of this cause of cardiac enlargement will be found under the head of hypertrophy of the organ.

The dilatation of the heart associated with exposure, unhealthy hygienic surroundings, insufficient or bad feeding, etc., is usually found among the poorer classes, and, as a rule, can be easily cured by the rest, warmth, and wholesome diet which is to be obtained in a hospital.

The treatment may, if necessary, be supplemented by the administration of tonics of various kinds, among which iron is usually the most suitable, given in combination with arsenic, strychnine, or phosphorus.

Cardiac tonics are seldom required, and in the absence of some special indication need not be prescribed.

A similar mode of treatment is applicable to those cases of dilatation of the heart which are dependent on anæmia, either simple or pernicious.

Malnutrition of the heart in common with the rest of the organs of the body is due to a variety of other causes, as, for instance, over-feeding, insufficient exercise, intemperance in the use of alcohol and tobacco, sexual excesses, and the like.

The cardiac dilatation which may be observed under such conditions is curable only when the patient is ready and able to give up his indulgences, and is prepared to regulate his habits and mode of life in accordance, so far as practicable, with the dictates of health.

A plain and spare diet should be ordered, and the bowels must be kept freely open by means of saline aperients and the occasional use of a brisk mercurial purge.

Hæmatinics are frequently of service, and they may be advantageously combined with digitalis, or with other cardiac and general tonics.

It is sometimes advisable to preface exercise by a few weeks complete rest in bed, during which time tissue change may be promoted and nutrition improved by the assistance of massage and passive movements of the limbs.

Exercise must be cautiously and gradually resumed, and at first may take the form of easy walking on level ground. Later the amount of exercise must be regulated by the requirements of each individual case.

It may be laid down, as a general rule, that any exercise which does not give rise to shortness of breath may be indulged in with safety and benefit.

A brief reference must be made to one or two of the special methods employed in the treatment of dilatation of the heart from failure.

The Schott treatment, which has of late years attracted very favourable notice, comprises the use of specially medicated baths, in conjunction with a carefully graduated series of resisted muscular movements. Although usually practised together, either procedure may be employed alone. This mode of treatment appears to be applicable to failure of the heart from any cause, but it is particularly adapted to those forms of dilatation which depend upon structural and nutritional lesions of the myocardium. (See p. 240.)

From the results so far obtained it would seem that the treatment is likely to occupy a permanent and prominent place in cardiac therapeutics.

Slight degrees of dilatation of the heart are sometimes greatly benefited by the system of graduated hill climbing at high altitudes, devised by Certe! The amount of food and water that is taken is also carefully regulated and restricted. This method of treatment is open to the objection that it is difficult to regulate the amount of exertion performed by the patient, and, unless great care is observed, strain is imposed on the heart, which may be productive of disastrous results.

The treatment of dilatation of the heart due to over-exertion consists in the removal of the cause, and the observance of complete rest for some weeks at least.

Exercise must be resumed gradually and cautiously, and any thing like severe or sudden exertion must be carefully avoided for many months.

Dilatation of the heart dependent upon acute intercurrent disease must be treated on the lines which were indicated when speaking of the maintenance of hypertrophy under similar circumstances.

Treatment of the effects.—The ultimate effects of dilatation of the heart from failure are seen both on the arterial and on the venous side of the circulation, and lead in the one case to anæmia, and in the other to engorgement.

Arterial anæmia is the outcome of the defective driving power of the ventricles.

Venous engorgement is the result of the obstruction to the outflow of blood from the great veins, consequent on the high pressure that obtains in the right auricle, which in turn depends on the general increase of intra-cardiac and pulmonary tension, working backwards through the tricuspid opening by way of the left heart, lungs, and right ventricle.

The object of treatment, therefore, is to increase the power and diminish the work of the heart, and at the same time to relieve cardiac distension and venous engorgement, to alleviate pain and distress, and to promote nutrition.

The attempt to fulfil these indications must be made in the first

instance from the venous side of the circulation, since it is easier, safer, and more effective to relieve the heart of its arrears of work than it is to increase the force of its contractions.

The relief of cardiac distension and venous engorgement, and of the work done by the heart.—Venesection affords the most speedy and effectual means of relieving distension of the right heart.

Except, however, in cases of acute dilatation this method of treatment is seldom practised, by reason of the inability of the enfeebled cardiac muscle to respond to any sudden form of relief. All that is necessary in the way of removal of blood can, as a rule, be more safely and conveniently effected by means of the application of eight or ten leeches over the præcordial or, preferably, the hepatic region.

The relief afforded by this procedure is usually very remarkable, and is, moreover, quickly produced.

At the same time, free purgation must be obtained, with the object of removing or, at least, of mitigating the engorgement of the liver and portal circulation, and of diminishing arterio-capillary resistance, and therefore of relieving the work of the heart.

For this purpose two or three grains of calomel may be given in combination with thirty or forty grains of compound jalap powder, which is by far the most useful of the hydragogue cathartics in cases of cardiac failure.

The bowels may be subsequently regulated by the use of one or two grains of blue pill, calomel, or grey powder with colocynth and hyoscyamus, which can be taken at night, once or twice a week, and followed the next morning by a mild saline aperient.

The action of the kidneys will be promoted by these measures, and, if necessary, may be further encouraged by the administration of cardio-vascular diuretics and direct renal stimulants, such as spiritus ætheris nitrosi, scoparium, and the like.

The salts of potassium are particularly useful as diuretics, on account of their valuable eliminative properties, and the acid tartrate possesses the additional advantage of acting on the bowels as a hydragogue saline purgative.

If the measures already mentioned, in conjunction with those about to be described, fail to afford sufficient relief to the venous congestion of the portal and systemic circulations, further assistance must be rendered to the heart by the direct removal of fluid from the œdematous tissues, and by drainage of ascitic accumulation, or pleural effusion.

These operations are most conveniently and safely performed by means of Southey's tubes, and should be carried out with the utmost care and under strict antiseptic precautions.

The relief afforded to the heart by the removal of even a small quantity of fluid in any of these situations is sometimes remarkable, and is often sufficient to turn the tide of events in the direction of recovery.

The work of the heart, which is of course greatly reduced by complete rest in bed, may be still further lightened, during an emergency, by the use of direct arterial dilators, such as the nitrites of amyl and sodium, nitro-glycerine, erythrol tetranitrate, and the spirits of nitrous ether.

The increase in the contractile power of the heart.—Acute primary dilatation, or urgent failure of the heart from any cause, should be met by the subcutaneous injection of ether, alcohol, or strychnine, which is the most rapid and potent means of cardiac stimulation that we possess.

Less serious cases may be treated by the internal administration of half-drachm doses of ether and sal volatile, in combination with four or five minims of liquor strychninæ, or alcohol may be given in the form of whisky or brandy.

The effect of these remedies can be supplemented by the simultaneous application of hot turpentine stupes or mustard poultices to the præcordium, or of ammonia or other pungent substance to the nose.

Under ordinary circumstances, however, the first indication in the treatment of cardiac failure is to relieve the heart of work, and this must be done in the manner indicated in the previous section.

The systole of the organ can then be strengthened by the use of cardiac tonics, which may when necessary be prescribed, at first, with stimulants such as strychnine, carbonate of ammonium, or spirits of nitrous ether.

Later they may be advantageously combined with iron, arsenic, or nux vomica.

The most important of the cardiac tonics are digitalis, squill, senega, strophanthus, convallaria majalis, sparteine, and caffeine.

Digitalis is the most powerful as well as the most generally useful of these remedies, and, with few exceptions, can be given in all forms of failure of compensation. It may be used alone, but it is often more efficacious when combined with squill or caffeine.

It is worthy of notice that the action of the cardiac tonics is frequently more powerful when two or three are given together than when the same drugs are prescribed separately and successively.

The administration of digitalis or of any of its allies should always be preceded by a purge, preferably in the form of two or three grains of calomel or blue pill, with half a drachm or more of compound jalap powder.

Next to digitalis the most useful cardiac tonic is strophanthus. Given alone, or in combination with convallaria majalis, it forms a very useful substitute for digitalis when the latter drug disagrees or has failed to do good.

The use of squill and senega as cardiac tonics is indicated when an expectorant effect is also desired, in which case either or both of these remedies may be exhibited in combination with nux vomica,

carbonate of ammonium, and other cardiac stimulants, or with other expectorants.

All the drugs which exert a tonic influence on the heart act, in addition, on the kidneys as cardio-vascular diuretics, and by promoting a free flow of urine they assist still further in relieving venous engorgement, and in promoting the absorption of dropsical effusion.

The alleviation of pain and distress and the promotion of nutrition.—No part of the treatment of heart failure requires more careful consideration than the use of sedatives, and there are no means which contribute so largely to the comfort and well being of the patient as the skilful and proper management of these remedies. On the other hand, incalculable harm may accrue from their indiscriminate administration.

Palpitation and præcordial pain may often be relieved by the local application of hot turpentine stupes, a few flying blisters, or a belladonna plaster. The internal administration of belladonna, bromide of ammonium, or small doses of opium in the form of Dover's powder, or the tincture, may be used for the same purpose.

Continuous dyspnoea or orthopnoea is sometimes greatly benefited by the subcutaneous injection of a small dose (gr. $\frac{1}{6}$ to $\frac{1}{3}$) of morphia. The inhalation of oxygen may also be of service in this respect.

Attacks of so-called cardiac asthma may be treated by free stimulation and the inhalation of nitrite of amyl. In severe cases relief can be obtained by the subcutaneous injection of morphia.

Nitro-glycerine, in doses of one-hundredth of a grain, three times a day, or erythrol tetranitrate, in half-grain doses, once or twice a day, may be given in the interval between the attacks.

Insomnia due to high arterial tension is relieved by the administration of one or two grains of calomel at night. In some instances sleep cannot be obtained except in the semi-upright position, and when this is the case much can be done to make the patient comfortable by the use of a suitable bedchair, or other means of support.

The venous engorgement and concurrent catarrh of the stomach and intestines greatly interfere with the processes of digestion and assimilation, consequently the feeding of the patient is usually a matter of considerable difficulty.

Small quantities of easily digestible food, such as milk, soup, beef-tea or beef-jelly, meat extract or raw meat juice, and raw eggs beaten up with brandy, may be given every three or four hours during the day and night. The taste of raw meat juice, which is a particularly valuable form of nourishment, may be disguised by jam or gravy.

There is no objection to the use of solid food provided the patient is able to digest it without discomfort.

If a sufficient quantity of nutriment cannot be taken by the mouth, the amount may be supplemented by rectal alimentation.

For this purpose two ounces of fresh or peptonised milk should be shaken up with an equal quantity of beef-tea and a raw egg, and the mixture is then slowly and gently injected into the bowel once or twice a day.

Fluids should be taken sparingly, more especially at meals. Thirst may be assuaged by sipping toast-water, or the preparation known under the name of "imperial drink," which contains the acid tartrate of potassium as its principal ingredient.

Alcohol in some form is usually required, but the amount must be strictly limited by the requirements of each individual case. It is seldom or never necessary to prescribe more than ten ounces of brandy or whisky in the twenty-four hours.

SECTION II

ACUTE MYOCARDITIS

ÆTIOLOGY

Acute inflammation of the wall of the heart is of very rare occurrence as a primary disorder, and when apparently observed as such its mode of origin is usually obscure, though chill and over-exertion have in some instances been regarded, with doubtful accuracy, as the exciting causes.

A few of the recorded cases have followed injury to the chest wall over the region of the heart.

With these exceptions, acute myocarditis arises in the course of some general disorder, such as acute rheumatism, and is then commonly, though not always, found in association with endocarditis or pericarditis, by reason of the direct extension of the inflammatory process to the wall of the heart, or as part of a general "carditis."

It sometimes occurs in relation with other acute febrile diseases, as, for instance, diphtheria, anthrax, enteric and scarlet fevers.

A large proportion of the cases are observed in connection with pyæmia due to acute osteomyelitis, and other suppurative affections of bones and joints, puerperal fever, malignant endocarditis, and the like.

Pyæmic affections of the cardiac walls are most commonly found in male subjects under the age of seventeen years. Thus out of fourteen cases collected by Sir Richard Quain, twelve occurred in boys, and in eleven of these the age of the patient did not exceed the limit just mentioned.

Myocardial inflammation is occasionally due to embolism of the branches of the coronary arteries, and in very rare instances it has been associated with malignant disease of the wall of the heart.

PATHOLOGICAL ANATOMY

Two forms of acute myocarditis are recognized, according as the inflammatory process is more or less diffused and terminates in fibrosis, or is circumscribed and results in the formation of abscess.

The part of the heart involved, in either event, is most commonly the wall of the left ventricle, and in the large majority of cases inflammation of the endocardium, or pericardium, or of both these membranes, coexists with the myocardial lesion.

In the diffused form of the disease the affected portion of the myocardium, which, at first, is of a deep red or purple hue, subsequently becomes reddish grey in colour, and finally presents a white, glistening, tendon-like appearance. During the early stages of the process the muscle fibres are swollen and softer than normal.

On microscopical examination the intermuscular septa are seen to be infiltrated with leucocytes, proliferated connective tissue cells, and sero-fibrinous exudation. The blood vessels are distended and engorged with blood, and hæmorrhages are not infrequently observed. The muscle cells are swollen and tend to split up transversely, while the normal striation of the fibres is indistinct or altogether lost on account of granular and fatty degeneration and nuclear proliferation.

The subsequent organization of the inflammatory products leads to further compression and atrophy of the muscle fibres, which become wholly or partially replaced by bands or patches of dense fibrous tissue.

In rare instances the muscular tissue has been found either exclusively or predominantly affected by inflammation, and to this variety of the disease the term parenchymatous myocarditis has been applied.

Purulent myocarditis usually, though not invariably, arises in connection with pyæmia, and appears in the form of yellowish white or greyish spots, situated immediately beneath the endocardium, or pericardium, or scattered irregularly throughout the wall of the heart.

A zone of hyperæmia surrounds each focus of inflammation, which subsequently develops into an abscess containing pus cells, granular and fatty debris, broken-down muscular tissue, and micro-organisms of various kinds.

The process of abscess formation, apart from pyæmia, differs in no respect from that already described.

General suppuration of the heart is of very rare occurrence, but it is not uncommon to find a widespread parenchymatous degeneration of the muscular substance of the organ in association with the ordinary form of the disease.

PATHOLOGY

The results of acute myocarditis depend on the nature, intensity, and extent of the inflammatory process.

In the diffused form of the affection, extensive implication of the myocardial substance may give rise to acute dilatation of the heart, while more severe and limited disease may be followed by local bulging of the wall of the organ, and the consequent formation of acute cardiac aneurism.

Incompetence of the auriculo-ventricular valves may be due to inflammation of the basal muscular ring surrounding these orifices.

Abscess of the heart may burst inwardly into the ventricles or auricles, or outwardly into the pericardial sac.

In the first event the contents of the abscess cavity gain access to the circulation, and may be the source of general pyæmic infection.

Rupture of the abscess into the pericardial sac is followed by acute suppurative pericarditis.

In non-pyæmic cases the abscess may burst both in an inward and outward direction, leading to rupture of the heart and fatal hæmorrhage into the pericardial cavity. A communication between the ventricles or auricles may be established by the rupture of an abscess situated in the interventricular or interauricular septum.

SYMPTOMS

The symptoms of acute myocarditis are usually more or less completely masked by those of the disease with which the myocardial lesion is associated. Moreover, when symptoms referable to implication of the muscular substance of the heart are observed, they can generally be more easily accounted for by the coexistence of inflammation of the pericardium or endocardium.

In well-marked, uncomplicated cases the patient is extremely restless, and complains of præcordial pain and uneasiness, accompanied by dyspnoea, palpitation, and irregular tumultuous action of the heart.

There is usually great muscular weakness and tremor. The expression is anxious, the face and lips may be pale or cyanosed, and the extremities are frequently cold and livid.

Attacks of giddiness or faintness, nausea, vomiting or sweating, may severally or collectively arise during the course of the disease.

The fatal termination may be preceded by headache, delirium, coma, and convulsions, or by the signs and symptoms of rapid failure of the heart. Sudden death is sometimes observed as the result of syncope or rupture of the heart.

PHYSICAL SIGNS

The physical signs of acute myocarditis are to a great extent obscured by those of concurrent inflammation of the endocardium or pericardium. If, however, these complications can be excluded the impulse of the heart is found to be feeble and diffused, or altogether impalpable. The area of cardiac dulness is increased chiefly in the lateral direction. The sounds of the heart are short and weak, and the first sound tends to become gradually fainter, so that finally it may cease to be audible. Murmurs may be heard in different situations, but they present no special features, except in cases of perforation of the interventricular septum, when a bruit of very variable character is suddenly developed, which is audible, as a rule, over the greater portion of the præcordial area.

The pulse is rapid and irregular, both in force and frequency, while the wave is small, short, and badly sustained.

DIAGNOSIS

The existence of acute myocarditis is usually more a matter of conjecture than of actual demonstration. If, as is rarely possible, the presence of both endocarditis and pericarditis can be excluded, the signs and symptoms of rapid failure of the heart, in association with acute rheumatism, would justify a diagnosis of myocardial inflammation. Moreover, when endocarditis or pericarditis are observed, the existence of myocarditis may be reasonably suspected, if the degree of cardiac disturbance is greater than can be fairly accounted for by the inflammation of the endocardium or pericardium, as the case may be.

The diagnosis of purulent myocarditis is even more difficult than that of the simple form of the disease.

The sudden appearance of a murmur indicative of rupture of the interventricular septum, followed by the signs and symptoms of general pyæmic infection, would afford strong evidence of the existence of cardiac abscess. The signs of valvular rupture, accompanied by a similar train of events, would not have the same significance, since this occurrence may likewise be due to malignant endocarditis.

In pyæmic cases the rapid development of cardiac failure, with or without the signs of acute pericarditis, might lead to a suspicion of myocardial inflammation.

PROGNOSIS

While there can be no doubt that recovery can and does take place after slight or moderate degrees of myocarditis, it is equally

certain that the diagnosis of the lesion, under such circumstances, is seldom or never made, with anything like precision, during life.

On the other hand, if the intensity or extent of the myocardial inflammation is sufficient to give rise to unmistakable evidence of its existence, the outlook is most unfavourable.

In cases of an intermediate character, the prognosis rests on the degree of impairment of the muscular power of the heart, which must be gauged by a careful consideration of all the symptoms and physical signs indicative of cardiac insufficiency.

Suppurative myocarditis is almost invariably fatal within a few days, though in some exceptional instances recovery has been accounted for by the inspissation and subsequent calcification of the purulent exudation.

In attempting to forecast the issue of acute myocarditis, the possibility of sudden death from rupture of the heart must be taken into account.

TREATMENT

The treatment of acute myocarditis consists mainly in the removal of the cause; the observance of complete rest; the alleviation of cardiac pain and excitement; and the maintenance of the strength of the heart.

The use of salicylate of soda in the treatment of acute rheumatism, complicated by inflammation of the myocardium, requires the utmost caution, on account of the depressing influence exerted by this drug on the heart. Salicin is less objectionable, but both these remedies may generally be replaced with advantage by quinine and alkalies, which can be given together in an effervescing mixture.

In pyæmic cases antiseptics may be tried, but in the light of recent research it would seem preferable to employ the hypodermic injection of antistreptococcus serum.

Absolute rest in bed should be insisted upon, and the patient must on no account be allowed to sit up, or even to raise himself. Cardiac irritability may be allayed by the application of belladonna, or warm fomentations to the præcordium, and the internal administration of belladonna, with digitalis or strophanthus.

Light digestible food in the form of milk, broth, beef-tea, or beef-jelly, raw meat juice, etc., must be given in small quantities at frequent intervals.

Stimulants, such as alcohol, ether, ammonia, and strychnine, and cardiac tonics are usually imperatively called for, but their use must be carefully adjusted to the requirements of the heart, since overstimulation of the organ might lead to rupture of the softened walls.

SECTION III

DEGENERATIVE DISEASES OF THE MYOCARDIUM

With the exception of parenchymatous, fatty, and fibroid affections, the degenerative diseases of the muscular walls of the heart, which comprise changes of a pigmentary, amyloid, hyaline, and calcareous nature, possess little or no clinical interest.

The precise pathological character of the morbid process described here as parenchymatous degeneration, but which is also known under the names of granular or albuminous degeneration, cloudy swelling, and parenchymatous or infectious myocarditis, has not yet been satisfactorily determined. By some it is regarded as a purely degenerative change, a view shared in by the author of this work, while by others it is considered to be of an inflammatory nature.

The mode of origin of the condition and its clinical significance will now be described in detail.

PARENCHYMATOUS DEGENERATION OF THE HEART**ÆTIOLOGY**

This form of degeneration affects chiefly the muscular and epithelial tissues, and is most commonly found in the course of acute febrile disorders, such as enteric, scarlet, and relapsing fevers, diphtheria, small-pox, typhus, erysipelas, septicæmia, and pyæmia. It also occurs in connection with pneumonia, acute rheumatism, and other conditions which give rise to long-continued pyrexia.

PATHOLOGICAL ANATOMY

The heart is usually more or less dilated, and the muscular tissue of the organ, which is diminished in consistence, doughy to the touch, and more friable than normal, presents on section a peculiar dull greyish yellow tint. The endocardium and pericardium may show a similar change of colour.

Under the microscope the normal striation of the affected muscle fibres is seen to be partially or wholly obscured by a finely granular deposit, which is irregularly distributed throughout the walls of the heart. By treatment with a few drops of glacial acetic acid, or a solution of potash, the granular material is dissolved and, except that they remain somewhat swollen, the muscle fibres resume an almost natural appearance.

Nuclear proliferation of the muscle cells and intermuscular connective tissue is frequently observed, and the blood vessels in the affected areas may be congested.

PATHOLOGY

The exact mode of production of this form of myocardial degeneration is still a matter of doubt. It has been attributed to the operation of the pyrexia, or of the special poison associated with the different ætiological conditions, on the muscular tissue of the heart. It has also been regarded as a post-mortem change dependent on some hitherto unexplained form of tissue alteration during life.

In whatever manner produced, parenchymatous degeneration is frequently, though by no means invariably, the immediate precursor of a fatty transformation of the myocardium. The two conditions may, however, be easily differentiated, since fatty disease is unaffected by the reagents mentioned above, whereas it is easily dissolved by chloroform and ether, which exercise no visible effect on parenchymatous degeneration. Moreover, the deposit is larger and more globular in the event of fatty change, and is stained black by osmic acid, a reaction which does not obtain in the case of the parenchymatous affection.

Parenchymatous degeneration of the myocardium seriously weakens the muscular power of the heart, consequently the presence of the condition is a factor of great weight in the prognosis of the disease with which it is associated.

SYMPTOMS AND PHYSICAL SIGNS

The symptoms of parenchymatous degeneration of the muscular walls of the heart, though seldom well defined, are those of cardiac insufficiency, developing during the course of one of the acute specific fevers. They are, however, of little or no use in the diagnosis of the lesion, which depends mainly on the evidence afforded by the physical signs. A careful examination of the heart from day to day in the course of an acute febrile disorder, such as enteric fever, will reveal a gradually increasing feebleness of the cardiac impulse, which may ultimately be altogether imperceptible. The first sound becomes correspondingly shorter and weaker, and may finally disappear or be replaced by a soft systolic bruit.

The force of the pulse declines with the increasing enfeeblement of the muscular power of the heart, and in advanced degrees of the affection the beat may cease to be palpable at the wrist.

DIAGNOSIS

The diagnosis rests on the occurrence of the signs just described in association with one of the acute specific fevers mentioned under the head of "Ætiology."

PROGNOSIS

Parenchymatous degeneration of the cardiac muscle adds greatly to the gravity of the disease of which it is a complication. The

degree of danger corresponds with the extent and rapidity of the enfeeblement of the heart, which may be gauged by a careful examination of the condition of the pulse, cardiac impulse, and first sound.

TREATMENT

The occurrence of this form of myocardial degeneration calls for the use of alcoholic stimulants, the amount of which must be regulated in accordance with the degree of cardiac disability.

SECTION IV

FATTY DISEASE OF THE HEART

The term "fatty disease of the heart" includes two distinct pathological conditions, which are distinguished under the titles of "fatty infiltration" and "fatty degeneration" respectively. The mode of origin and significance of the two affections are essentially different, so that it will be necessary to consider them separately.

FATTY INFILTRATION

This disease is characterised by an increase in the amount of sub-pericardial fat, which oftentimes penetrates between the muscular fibres of the heart.

ÆTIOLOGY

Fatty infiltration of the heart is most commonly observed as a local manifestation of general obesity, but it is occasionally found in connection with wasting disorders, such as malignant and tuberculous affections.

The tendency to accumulate fat is sometimes hereditary, though more often it is acquired as the result of over-eating and over-drinking, in association with an insufficient amount of exercise. Fatty infiltration of the heart occurs more frequently among men than women, and is seldom met with before the age of fifty.

PATHOLOGICAL ANATOMY

In well-marked cases the heart lies embedded in a covering of fat, which extends more or less deeply into the muscular substance of the organ, and may even involve the columnæ carneæ and musculi papillares. The overgrowth of fat is accompanied by a corresponding amount of attenuation of the myocardium, so that finally the wall of the heart may appear to be almost wholly converted into adipose tissue. Under the microscope the pericardial

connective tissue is seen to be loaded with fat cells, which invade the myocardium along the lines of the intermuscular septa. The muscle fibres, more especially in the outer portions of the cardiac wall, are thinned out and separated by wide tracts of the fatty overgrowth. The course of the fibres may be variously twisted, but structurally they present, as a rule, a perfectly normal appearance. In very advanced cases, however, a true fatty degeneration of the muscular fibres may coexist with the fatty infiltration.

PATHOLOGY

The relation between the fatty hyperplasia and the atrophy of the muscle fibres is still uncertain, though there appears to be little doubt that it is not one of cause and effect. In all probability the muscular atrophy is the primary change, and is followed by the fatty overgrowth, but it may be that both events run concurrently and are the results of the same cause.

The accumulation of fat on the surface and between the fibres of the heart may be sufficient to seriously hamper the movements of the organ, but the chief source of cardiac weakness arises, no doubt, from the atrophy of the myocardium, which may be of considerable extent.

Great thinning of the muscular parietes may be followed by rupture of the heart.

SYMPTOMS AND PHYSICAL SIGNS

Slight degrees of fatty infiltration of the heart give rise to no symptoms or signs by which the disease can be recognised, with any certainty, during life.

The condition may, however, be suspected in a corpulent individual who suffers from præcordial uneasiness or pain, and shortness of breath on slight provocation, with, it may be, paroxysms of dyspnœa or attacks of faintness, and presents in addition the signs of some degree of cardiac dilatation.

The symptoms and physical signs of the advanced stages of the affection resemble those of fatty degeneration of the heart, and will be described under that heading.

DIAGNOSIS

The diagnosis of fatty infiltration of the heart rests on the occurrence of the symptoms and signs of cardiac insufficiency in a person of corpulent habit.

PROGNOSIS

In attempting to forecast the issue of fatty infiltration of the heart it is necessary to take into consideration the amount of damage sustained by the cardiac muscle, and the extent to which repair is likely, as well as the effects of treatment on the general obesity. So long as the myocardium is sound, and provided the general

excess of fat can be reduced by therapeutic and other means, the prognosis will be favourable. Extensive atrophy of the muscular parietes of the heart is of grave import, and in such an event the possibility of sudden death from syncope or rupture of the thinned walls must not be lost sight of.

TREATMENT

The objects of treatment are to remove or reduce the general accumulation of fat, to sustain the strength of the heart, and to improve the nutrition of the cardiac muscle.

The diet must be carefully regulated with the view of restricting the amount of starchy and fatty foods, and of maintaining a suitable proportion of proteids.

Strict moderation should be observed in the use of tobacco and alcohol, which, in the form of spirits and malt liquors, must be absolutely prohibited.

The patient should be encouraged to take moderate and regular exercise in the open air, and the action of the skin and bowels may be assisted by diaphoretics and mild saline aperients.

The circulation should be maintained in as tranquil a condition as possible, and all sources of cardiac strain and excitement sedulously avoided.

Graduated muscular exercise, such as is obtained under the Schott and Cœrtel systems of treatment, is sometimes of very great service.

The action of alkalis, and of the salts of iron and iodine, is reputed to be of service in the treatment of general obesity, and one of these remedies may be employed in combination with some general tonic, such as arsenic, strychnine, or quinine.

Cardiac tonics, more especially digitalis, must be given with caution, and the effects should be carefully noted.

The incidence of acute intercurrent disease is an indication for the liberal use of stimulants, on account of the usual inability of fat subjects to satisfactorily withstand shock or strain of any kind.

The use of so-called "anti-fat" remedies, and of thyroid extract in the treatment of obesity is not unattended by danger.

FATTY DEGENERATION OF THE HEART

The essential feature of this disease is a fatty transformation of the muscular substance of the heart.

ÆTIOLOGY

Fatty degeneration of the heart is due to interference with nutrition, more particularly with the process of oxidation, which may depend either on an impoverished or on an insufficient supply of blood to the myocardium, the result of general or local causes.

Accordingly, fatty degeneration of the heart is observed in con-

ditions of general anæmia, especially pernicious anæmia, and of poisoning by phosphorus, and it may be by arsenic, antimony, mercury, and lead. It is also found in connection with long-continued pyrexia, and the puerperal state, and occasionally with chronic cachectic conditions accompanying prolonged suppuration and wasting disorders.

Interference with the circulation through the coronary arteries constitutes the most important local cause of fatty degeneration of the heart. This may be due to atheromatous disease of the coronary arteries, or of the aorta involving the orifices of these vessels, or it may depend on failure of the left ventricle from any cause.

The fatty degeneration of the cardiac muscle fibres which may be observed in cases of enlargement of the heart is occasioned by one or other of these conditions.

Fatty degeneration of the heart occurs also in association with acute myocarditis, and more especially with the suppurative variety of this affection.

The general causes of fatty degeneration of the heart are usually operative before middle life, and in women rather than in men, while fatty degeneration due to coronary disease is rare before the age of sixty, and is found most commonly among men.

The mode of origin of fatty degeneration of the heart may be contrasted rather than compared with that of fatty infiltration, since the one is the result of under-nutrition, the other of over-nutrition.

PATHOLOGICAL ANATOMY

The morbid appearances vary according as the fatty change arises in connection with general causes, and is widely diffused through the wall of the heart, or depends on local conditions, and is concentrated at one or more spots.

In the former case the heart is usually somewhat enlarged, more often as the result of dilatation than of hypertrophy, but it may not exceed the average size, and is sometimes smaller than normal.

The muscular substance is soft and friable, and of a light brown colour, interspersed with greyish yellow streaks or patches, which correspond with the sites of the degenerative changes. The parts most affected are the musculi papillares and columnæ carneæ.

In typical cases these structures present a characteristic transverse striation, made up of alternate reddish brown and pale yellow markings, an appearance that has been likened to the fur of a tabby cat. This peculiar mottling seen through the endocardium is not observed on section of the muscle, the cut surface of which exhibits a uniform yellowish brown or buff colour.

On microscopical examination the irregular distribution of the fatty change is more evident than it is to the naked eye. Fibres in all stages of degeneration may be seen running side by side

with others that are perfectly healthy. The morbid change appears at first in the form of minute globules of fat, collected at either end of the nucleus, or scattered in clusters throughout the substance of the muscle cell.

The process gradually extends until finally the whole structure of the fibre is completely effaced by small oil globules, which preserve a singular uniformity of size and shape. They are ultimately set free by the disintegration of the affected muscle cells.

In the local form of the disease the degenerative changes are confined to one or possibly more patches of variable size, situated usually in the wall of the left ventricle. The affected area is of a yellowish brown colour, breaks down easily under the finger, and may exude an oily fluid on pressure.

The microscopical appearances do not differ from those already described, except that relatively a larger number of fibres are affected by the morbid process.

PATHOLOGY

The effects of fatty degeneration of the cardiac muscle are to weaken the contractile energy of the heart, and to diminish either generally or locally the resisting power of its walls.

The result, in the case of the general form of the disease, is the production of a variable degree of dilatation of the organ, which may be attended by sudden and fatal syncope. This event does, however, sometimes happen in the absence of any enlargement of the heart.

Local weakness of the cardiac parietes may lead to aneurismal bulging, or to rupture of the wall of the heart, with fatal hæmorrhage into the pericardial sac.

The liability to sudden death from one of the causes just mentioned is greater in this form of heart disease than in any other.

The extent to which repair is possible in cases of fatty degeneration of the heart is not known, but there can be little doubt that slight degrees of the affection, as, for instance, those due to anæmia, can be completely recovered from.

SYMPTOMS

The symptoms of fatty degeneration of the heart, more especially of the early stages of the affection, are very indefinite, and it not infrequently happens that the disease is discovered post-mortem without any evidence of its existence having been detected during life. In such cases it may be that the symptoms due to implication of the heart are merged in those of the primary disorder, and thus escape observation, or that the organ, though crippled, is still equal to the ordinary requirements of the circulation, which,

under the circumstances, are usually below par. In the latter event a comparatively slight cause, such as moderate exertion or excitement, may be sufficient to produce arrest of the heart's action and sudden death.

During the early stages of the disease the symptoms are for the most part referable to cardiac insufficiency, and do not differ from those of commencing dilatation from other causes.

Thus the patient may suffer from præcordial pain, palpitation, and breathlessness on slight exertion, often accompanied by a feeling of tightness or constriction of the chest.

Attacks of faintness or giddiness are sometimes observed to follow even a moderate amount of muscular effort. Want of energy, headache, sleeplessness, or drowsiness may be complained of, and are indicative of disturbance of the cerebral circulation. As the disease advances the symptoms become more pronounced and less equivocal.

The shortness of breath increases and tends to become habitual, while attacks of cardiac asthma and angina pectoris may occur from time to time. The fatal issue may be preceded by the phenomenon known under the name of 'Cheyne-Stokes' respiration.

A point of considerable interest and significance is the usual absence of well-marked venous congestion or serious dropsy, though it cannot be said that this peculiar feature of the disease has been satisfactorily accounted for.

The final stages of fatty degeneration of the heart are characterised in some instances by syncopal attacks, which may terminate fatally, and by seizures of an epileptic and apoplectic nature. In the majority of cases death occurs suddenly from syncope, coma, or rupture of the heart.

PHYSICAL SIGNS

Physiognomy.—A white satiny or greasy condition of the skin has been described in association with fatty degeneration of the heart, but in a great many instances the patient presents a fairly healthy appearance, even up to the time of death.

The presence of arcus senilis is no longer considered to be of importance in the diagnosis of the disease.

Pulse.—The pulse is usually irregular in force and frequency, and this is especially noticeable after exertion. The radial artery is of variable size, and cannot, as a rule, be felt between the beats. The wave is small, sudden, short, and badly sustained. The pulse rate may occasionally be very frequent or very slow, a point of some diagnostic value in either event.

Heart.—The cardiac impulse is feeble and diffused, or altogether impalpable. The area of percussion dulness may be increased, chiefly

in the lateral direction; on the other hand, it is often of normal extent.

The first sound at the apex is short and weak, and may be almost inaudible in advanced stages of the disease. It is sometimes accompanied by a soft systolic murmur. The second sound, as compared with the first, is often clear and distinct.

Disturbance of the time relations between the sounds, when observed, is always in the direction of approximation.

DIAGNOSIS

There are no symptoms or physical signs which can be said to be characteristic of fatty degeneration of the heart. Consequently the diagnosis of the condition is usually a matter of very great difficulty, and is seldom made with much certainty.

The occurrence of the symptoms and signs of cardiac insufficiency after middle age, which cannot be accounted for by valvular disease or other obvious cause, would point to the existence of myocardial degeneration. This indication acquires additional significance when associated with a history of intemperance, or of malnutrition from other causes, and with the presence of atheromatous changes in the coats of the accessible arteries. Under circumstances of this kind, the appearance of symptoms indicative of serious cardiac inadequacy, which are unaccompanied by the signs of venous congestion and dropsy, would justify a diagnosis of fatty degeneration of the heart.

PROGNOSIS

The prognosis of fatty degeneration of the heart turns for the most part on the cause and extent of the morbid process. If the cause can be discovered and is removable, as may happen in cases of anæmia, long-continued pyrexia, and of poisoning by various substances, the prospect of recovery is good.

On the other hand, fatty degeneration of the heart due to obstruction of the coronary arteries is irremediable, and a fatal termination is sooner or later inevitable. In any case, the attempt to estimate the probable duration of the disease must be qualified by the liability to sudden death, which may be the result of syncope or rupture of the heart.

TREATMENT

Treatment must be directed to the removal of the cause and the improvement of the general health, while all sources of cardiac strain and excitement should be carefully avoided.

Malnutrition of the heart, due to anæmia or to impoverishment of the blood arising in other ways, calls for the use of hæmatinics and general tonics, the action of which may be supplemented by suitable hygienic and dietetic means.

The treatment of the myocardial changes associated with febrile conditions will be found under the head of parenchymatous degeneration of the heart.

In the local form of the disease, which is due to obstruction in the coronary circulation, little or nothing can be effected in the way of the removal of the cause, but much may be done to promote the comfort of the patient, and to prolong life.

It is of the first importance that all sources of cardiac strain, such as bodily or mental effort, excitement, anxiety, and especially sudden exertion, should, so far as practicable, be avoided.

Exercise on the level ground may be permitted, and will be beneficial, provided it does not give rise to pain or shortness of breath.

The patient must be warmly clad, and he should take care not to expose himself to cold.

Dietetic treatment consists in the use of light nutritious food, which should be taken in moderate quantities, and at regular intervals. Alcohol, tea, and coffee may be taken sparingly, while tobacco should be altogether prohibited.

A daily evacuation of the bowels is desirable, and should be effected by mild aperients. The use of strong purgatives for this purpose is not unattended by danger.

Treatment by drugs is seldom of much service, though the administration of general tonics, such as iron, arsenic, and strychnine, appears to do good in some cases. Benefit is occasionally derived also from the use of digitalis, but this drug and its allies must be employed with caution, on account of the increased strain which their action may impose on the weakened cardiac walls.

Dyspepsia and flatulence may be a source of discomfort and danger, and their occurrence, in spite of a careful regulation of the diet, must be met by prompt and vigorous treatment in the shape of stomachic carminatives and antiseptics in combination with nuxvomica and general stimulants.

Failure of the heart is an indication for the liberal administration of alcohol, in the form of whisky or brandy, with, if necessary, ether, ammonia, and strychnine.

In other respects treatment may be conducted on the lines laid down under the head of cardiac dilatation.

SECTION V

FIBROID DISEASE OF THE HEART

ÆTIOLOGY

Fibroid disease of the heart may depend on a diffused or circumscribed hyperplasia of the intermuscular fibrous tissue, or on a

localized necrosis of the cardiac wall (*myomalacia cordis*) with subsequent regeneration and fibrosis.

A variety of the general form of the disease, described by Sir Richard Quain under the title of "connective tissue hypertrophy of the heart," has been attributed to over-feeding. With this exception, a diffuse intermuscular fibrosis of the myocardium is found in association with long-continued venous congestion of the heart, or with disease of the kidneys.

A circumscribed fibroid change is sometimes the sequel of an acute interstitial myocarditis, or it may be due to the extension along the intermuscular septa of a chronic indurative process, that has originated in inflammation of the endocardium or pericardium.

In the majority of cases, however, circumscribed fibroid disease of the heart arises in connection with *myomalacia cordis*, which is the result of an anæmic necrosis, or, in other words, of infarction of the cardiac wall, consequent on obstruction of the coronary circulation.

This condition may depend on atheroma, endarteritis obliterans, or embolism of the coronary arteries and their branches, and is therefore most commonly observed among males in relation with old age, gout, alcoholism, chronic Bright's disease, syphilis, and prolonged muscular exertion.

Syphilitic disease of the wall of the heart occurs also in the form of a patchy interstitial fibrosis, which may be the result of a simple inflammatory induration of the myocardium, or of gummatous changes.

PATHOLOGICAL ANATOMY

A diffuse interstitial fibrosis of the myocardium is usually found in association with great enlargement of the heart, wherein hypertrophy and dilatation are variously combined. The walls of the organ, which may be thickened or thinned, are of a peculiar tough leathery consistence, and do not collapse on section. Under the microscope the intermuscular fibrous overgrowth may be observed in all stages of development, from small round and spindle cells to wavy bands of fibrillæ. The muscular tissue may present a perfectly normal appearance, or may be in some stage of granular or fatty degeneration.

The circumscribed form of the disease appears in the shape of streaks, bands, or patches of yellowish or greyish white fibrous tissue, situated in the substance of the myocardium, most commonly near the apex, or in some other portion of the wall of the left ventricle. Implication of the right ventricle is usually due to the extension of the morbid process from the left side of the heart.

The amount of fibrous material and its mode of distribution are subject to considerable variation. Thus it may lie deeply embedded in the muscular tissue of the heart, so that it is seen only on section of the organ, or it may replace more or less com-

pletely the entire thickness of the cardiac wall over a considerable area. In other instances it appears as a local opaque thickening immediately below the endocardium or pericardium, whence it radiates, in the form of streaks or bands, between the adjacent muscle fibres. At the same time, the endocardium or pericardium, or both these membranes, are commonly the seat of the chronic inflammatory changes. Adhesion between the two layers of the pericardium, whether general or partial, is always most dense and firm at the spot where the fibroid material reaches the outer surface of the heart, and is not infrequently limited to this situation.

Thrombosis may occur over the corresponding site on the endocardial surface.

A localized fibrosis of the papillary muscles is not uncommon, while in rare instances the entire column may undergo a fibroid transformation. This change has no relation with the fibrous thickening that is observed at the apices of these structures, in common with the valves and chordæ tendineæ, in cases of long-continued high intra-cardiac tension.

The wall of the heart over a fibroid patch is usually more or less thinned, but, on the other hand, an increase in thickness is sometimes found.

Under the microscope the normal structure of the myocardium, in the affected area, is seen to be largely replaced by dense fibrous tissue, containing few cellular elements. In the central portions of the diseased patch all trace of muscular substance may have disappeared, while, at the margins, the bundles of muscle fibres are separated by wide tracts of the fibroid growth. The atrophy of the muscle cells is not accompanied by any well-marked structural alteration, though granular and fatty changes are occasionally observed.

PATHOLOGY

The effects of fibroid disease of the heart vary with the extent and site of the morbid process. A general interstitial fibrosis of the myocardium must interfere more or less with the functional activity of the heart, and consequently is accompanied by hypertrophy and dilatation of the organ, though, for obvious reasons, the latter condition usually preponderates.

A like result may follow the circumscribed form of the disease, more especially if it is of considerable extent. On the other hand, a localized fibrosis of the myocardium, if small in amount, may exist without giving rise to any alteration in the size of the heart or even to any appreciable functional disturbance. Implication of the papillary muscles may be a cause of valvular incompetence.

An important effect of circumscribed fibroid disease is the local weakening of the cardiac parietes, which most commonly affects some portion of the wall of the left ventricle. The diseased area may give way before the intra-ventricular pressure, so that a local

bulging or aneurism of the wall is produced, which may mechanically interfere with the movements of the heart, or may lead to sudden death by the bursting of the sac. A communication between the two ventricles is sometimes due to the rupture of an aneurism of the interventricular septum.

Cardiac aneurism is of exceedingly rare occurrence, except as the result of fibroid disease of the heart. In some instances the fibroid material undergoes contraction, instead of expansion, with the production of a puckered cicatrix of the cardiac wall, or of narrowing of one or other of the orifices of the heart.

Annular contraction of the conus arteriosus, arising in this way, is known under the name of "stenosis of the heart."

SYMPTOMS

The symptoms of fibroid disease of the heart are very indefinite. In some instances the affection is unaccompanied by any appreciable functional disturbance of the organ, while in others the first and only indication of the existence of the lesion is sudden death.

The abrupt cessation of the heart's action under such circumstances has not yet been satisfactorily explained.

In the majority of cases, however, the symptoms of the disease are practically identical with those of cardiac dilatation and insufficiency due to other causes, and comprise dyspnoea on exertion, palpitation, and præcordial pain or distress, followed sooner or later by cough, hæmoptysis, and the signs of pulmonary congestion, general venous engorgement and dropsy.

Attacks of syncope, cardiac asthma, and angina pectoris are sometimes prominent features of the affection.

Fibroid disease of the heart generally runs a chronic course, and terminates with the usual signs and symptoms of cardiac failure. Sudden death is sometimes observed as the result of rupture of an aneurism of the heart, or of embolism of one of the coronary arteries.

PHYSICAL SIGNS

Physiognomy.—There is nothing characteristic about the appearance of patients suffering from fibroid disease of the heart, though a variable degree of cyanosis may be developed with the occurrence of failure of the organ.

Pulse.—The pulse is usually irregular, both in force and frequency, a feature that has been held to be of some diagnostic value. A very slow pulse rate, not exceeding thirty beats to the minute, is occasionally observed. In other respects the characters of the pulse do not differ from those associated with cardiac failure from other causes.

Heart.—Physical examination generally reveals the existence of enlargement of the heart, which as a rule is mainly due to dilatation.

Careful percussion might in some instances map out an area of dulness suggestive of cardiac aneurism. The first sound at the apex may be accompanied by a systolic murmur due to mitral incompetence, which may depend on dilatation of the left ventricle or on fibrosis of the papillary muscles.

DIAGNOSIS

The diagnosis of fibroid disease is at all times a matter of very great difficulty, and in the present state of our knowledge impossible in the majority of cases.

When it is possible to exclude other sources of heart weakness, such as valvular lesions and the extra-cardiac causes, the occurrence of enlargement and insufficiency of the organ in association with general arterial disease would point to the probable existence of fibroid degeneration of the myocardium. This indication would be supported by evidence of pericardial adhesion, or of syphilis.

PROGNOSIS

When the diagnosis of fibroid disease of the heart can be made, the prognosis is most unfavourable.

TREATMENT

Treatment can be palliative only, and should be conducted on the general principles described under dilatation of the heart.

All sources of cardiac strain and excitement should be sedulously avoided, and tranquillity of the circulation promoted by rest and other appropriate hygienic and dietetic means.

Digestive derangements should be promptly rectified, and the bowels carefully regulated.

Little benefit is derived from the use of drugs, though arsenic and iodide of potassium seem occasionally to be of service.

Digitalis must be prescribed cautiously, in view of the harmful effects that sometimes have been observed to follow the administration of the drug in cases of this kind.

In the treatment of heart failure, reliance should preferably be placed on cardiac stimulants, such as ether, alcohol, ammonia, and strychnine, which must be given in quantities suitable to the requirements of the occasion.

SECTION VI

GROWTHS IN THE HEART

Among the morbid growths which may attack the heart are carcinoma, sarcoma, tubercle, syphilis, lymphadenoma, myoma,

fibroma, lipoma, hydatid cyst, and cysticercus. All these conditions are rare, some exceedingly so, and are interesting from a pathological rather than from a clinical point of view. The more common and important growths only will be briefly considered.

MALIGNANT DISEASE

Both carcinoma and sarcoma are almost invariably secondary to similar lesions in other parts of the body, though a few instances of primary implication of the heart are on record. In some cases the organ is invaded by the direct extension of the growth from a primary focus in the mediastinum, lungs, or stomach.

Carcinoma occurs in the encephaloid, scirrhous, or epitheliomatous form, while sarcoma may be of the lympho-sarcomatous or melanotic variety.

The morbid growth, in either case, is usually multiple, and appears most commonly immediately beneath the endocardium or pericardium, though the substance of the myocardium is sometimes involved.

Implication of the endocardium or pericardium may be associated with inflammation of these structures.

The right side of the heart is said to be more commonly the seat of malignant disease than the left.

TUBERCLE

Tubercular disease of the myocardium apart from a similar affection of the pericardium is exceedingly rare. Miliary tubercles have been observed in the intermuscular connective tissue in cases of acute general tuberculosis. In other instances the disease has appeared in the form of sub-pericardial caseous nodules.

HYDATID CYST

The heart is sometimes the seat of hydatid cysts, which may be single or multiple, and are situated in the substance of the myocardium on either side of the organ. The tumour may project either inwards or outwards, and may rupture in either or both of these directions. In other cases the sac may lead to obstruction of one or other of the cardiac orifices, or to incompetence of the valvular structures.

SYPHILIS

The manifestations of this disease with respect to the heart have already been referred to under fibroid degeneration of the myocardium.

LYMPHADENOMA

The heart is sometimes involved, in common with the other organs of the body, in cases of general lymphadenoma.

Non-malignant tumours of the heart are exceedingly rare, and possess no clinical interest.

SYMPTOMS

In a large number of the cases of cardiac neoplasmata no symptoms have been present. Malignant disease of the heart has occasionally been associated with severe præcordial pain. When the lungs are affected by the morbid growth as well as the heart, cough, hæmoptysis, and dyspnœa are prominent symptoms.

PHYSICAL SIGNS

Evidence of pericarditis is sometimes obtained in cases of malignant and tubercular disease of the heart. In other instances the tumour gives rise to obstruction of an orifice or to incompetence of a valve, and in this way may be accompanied by the signs and symptoms of valvular disease.

DIAGNOSIS

The diagnosis, which is hardly ever made, rests on the occurrence of the signs and symptoms of cardiac insufficiency in connection with the presence of a new growth in some other portion of the body.

PROGNOSIS

If it were possible to make a diagnosis of a new growth in the heart, a fatal prognosis only could be given.

TREATMENT

Treatment consists in the relief of pain and distress, which may be effected by the measures indicated in previous sections of this work.

CHAPTER XXIII

ANGINA PECTORIS

Ætiology—Kinds of Angina—Morbid Anatomy—Pathogenesis—Symptoms—
Physical Signs—Diagnosis—Prognosis—Treatment.

“ANGUISH of the heart” is, perhaps, the best paraphrase of this term.

ÆTIOLOGY

In the present state of our knowledge of angina pectoris it is difficult to give a systematic account of the ætiology, morbid anatomy, and pathology of this condition, but an attempt, which must be regarded as tentative, will be made to consider the disease under these heads.

Angina pectoris in the form originally portrayed by Heberden is a rare disorder. It occurs, in the large majority of cases among men, between the ages of forty and sixty-five. Statistics show that the proportion of men to women attacked by the disease is about as ten to one.

The incidence of angina pectoris exhibits a marked hereditary tendency.

Physical and mental strain, gout, rheumatism, syphilis, and plumbism are ætiological factors in so far as they are concerned in the production of cardio-vascular disease.

Any of the conditions (see p. 79) which raise arterial tone, and thereby increase arterio-capillary resistance, also act as contributory causes of angina pectoris.

The abuse of alcohol and tobacco exerts a causal influence in that the action of these drugs in excess gives rise not only to nervous disturbance of the heart and vaso-motor mechanism, but also to organic disease of the cardio-vascular system.

A certain number of cases of angina pectoris have been observed to follow influenza.

The chief exciting cause of the attack is physical exertion. A comparatively small muscular effort, more especially when made suddenly, is oftentimes sufficient to bring on a paroxysm. Emotional disturbance, particularly in the form of anger or nervous shock, is

also a potent means of exciting an attack. Among other important proximate causes of the disease are indigestion with flatulent distension of the stomach, constipation, and cold applied to the surface of the body, *i.e.* by cold winds, or by bathing.

Two forms of angina pectoris are recognised, viz. :—

1. True angina pectoris
2. False angina pectoris

So far the ætiology of true angina pectoris only has been considered.

False angina pectoris, or pseudo-angina pectoris, or angina pectoris vasomotoria, as it is now commonly designated, is subdivided into two groups, the neurotic and the toxic.

The neurotic variety occurs at any age, but is most prevalent between puberty and middle life. Women are rather more commonly affected than men. An hereditary tendency is well marked.

Occupations involving nervous strain predispose to the occurrence of this form of angina through the vaso-motor disturbance to which they so frequently give rise.

Any of the conditions which lead to increased arterio-capillary resistance may be connected with the causation of angina pectoris vasomotoria.

Influenza has appeared to exert a causal influence in some cases.

The toxic form of pseudo-angina is ascribable to the abuse of tea, coffee, tobacco, etc.

Huchard distinguishes three forms of tobacco angina, viz. :—

1. A form due to functional spasm of the coronary arteries, produced by the direct action of the drug on the vaso-motor mechanism of the heart.
2. A form due to arterial spasm consequent on the dyspepsia produced by tobacco.
3. A form due to arterio-sclerosis with narrowing of vessels, produced by chronic tobacco poisoning.

One of the most important exciting causes of an attack of pseudo-angina, in all its forms, is chilling of the surface of the body, which may be brought about by exposure to cold winds, by immersion in cold water, by insufficient covering during sleep, etc. Mental emotion, physical exertion, dyspepsia with flatulence and constipation, are also frequent exciting causes of pseudo-angina.

MORBID ANATOMY

True angina pectoris is associated with a great many different morbid conditions of the heart and vessels, which may be briefly classified as follows:—

1. **Disease of the endocardium.**—Aortic incompetence is the valvular affection most commonly found. Aortic stenosis is, however, not infrequently observed, and in very rare instances mitral stenosis has been noted.

2. **Disease of the pericardium.**—Adherent pericardium is occasionally present, but always in association with aortic valvular disease.

3. **Disease of the myocardium.**—Fatty infiltration of the cardiac wall is sometimes found independent of disease of the coronary vessels.

In the great majority of cases, however, lesions of the cardiac wall depend on arterio-sclerosis (atheroma) of the coronary arteries and their branches, or on aortic disease involving the orifices of one or both of these vessels.

The coronary arteries and their branches become partially or wholly occluded, or converted into rigid and thickened calcareous tubes, with a corresponding degree of interference with the blood supply to the wall of the heart.

The more important morbid changes which are produced by interference with the coronary circulation are fatty infiltration and fatty degeneration of the cardiac wall, fibroid or fatty fibroid disease of the heart, and hæmorrhagic infarction or softening of the cardiac muscle.

The heart is usually enlarged, and its walls soft and flaccid.

4. **Disease of the aorta and systemic arteries.**—The aorta is commonly the seat of atheromatous disease, which may involve the orifices of the coronary arteries.

Aortitis usually gives rise to angina. Dilatation of the ascending portion of the aorta is frequently found, and in some instances there is an aneurism of the vessel. The systemic arteries commonly show arterio-sclerotic (atheromatous) changes.

5. **Disease of the cardiac plexuses and ganglia.**—In a few instances pigmentary, granular, and other degenerative changes have been found in the vagi, cardiac plexuses, and cardiac ganglia.

It is stated that in some fatal cases of angina pectoris a careful examination of the heart and vessels has failed to reveal any sign of disease.

Pseudo-angina pectoris has no morbid anatomy.

PATHOLOGY

Since the morbid conditions of the heart and systemic arteries enumerated above are found much more commonly without than with the symptoms of angina pectoris, it is clear that they operate as contributory causes only of the attack.

The central, indeed the essential, feature of angina pectoris is the inability of the heart for the time being to adequately perform its work, and it is from this standpoint that the pathology of the two varieties of the disease can be profitably compared and harmonized.

In angina pectoris, of whatever variety, the inability of the heart to cope with its work may depend on—

1. Mural weakness (absolute or relative).
2. Increased peripheric resistance.
3. A combination of mural weakness and increased peripheric resistance.

Now, the morbid changes found in the cardiac wall in connection with angina pectoris are not evenly distributed throughout the heart. In other words, the areas of disease and the lesions of the coronary vessels, or their branches, are scattered irregularly throughout the wall of the ventricles, auricles, and aorta.

It may be pointed out also, that the nervous tissue (*i.e.* ganglia and nerve fibres) distributed over the wall of the heart probably suffers in common with the muscular substance of the organ.

If, now, stress be thrown on the heart, the areas of diseased wall are unable to take their share in resisting the increased intra-cardiac pressure, and consequently become unduly stretched and distended.

Again, a coronary artery, or one or more of its branches, is the seat of atheromatous disease, whereby the vessel or vessels become thickened and rigid, or partially occluded.

The areas of distribution of the affected vessels may be sufficiently supplied with blood so long as the heart is acting quietly. So soon, however, as stress is imposed on the organ the diseased vessels are unable to satisfy the additional demand for blood, and the muscle fibres in the areas supplied are thus temporarily thrown out of action, and in consequence become unduly stretched and distended by the increased intra-cardiac pressure.

The pain felt under these circumstances is accounted for by the stretching and tension of the nerve elements in the areas involved, and it has already been pointed out that these structures are, in all probability, unusually impressionable and irritable.

The distribution of the pain is explained by the connection of the sensory nerves of the heart and aorta with the spinal cord from the third cervical to the eighth dorsal roots. The overflow of sensory nerve stimuli from the heart appears to travel chiefly along the

second dorsal roots, which are in connection with the left ventricle, where presumably the pain most commonly originates.

The sense of impending death which is a characteristic feature of a fully developed attack of angina pectoris is readily explained by the hypothesis under consideration. It is obvious that if distension and stretching of localized areas of the cardiac wall take place during systole the ventricular parietes are simultaneously undergoing contraction and expansion. The effect of this dynamical perversion of the cardiac movements on the impressions transmitted to the cardiac centre during the ventricular contraction must be contradictory and antagonistic. The cardiac centre must under these circumstances be thrown into a turmoil, and herein lies the explanation of the sense of impending death. The disturbance of the cardiac centre spreads to adjacent centres whereby it becomes possible to explain the nausea, flatulence, hiccough, etc., that are so commonly associated with the anginal paroxysm.

The occurrence of sudden death is explicable on the assumption that the arrival at the cardiac centre of equally balanced antagonistic impulses inhibits the action of the centre.

The view advanced here will explain the occurrence of the so-called angina pectoris sine dolore (Gairdner) as an occasional mode of termination of true angina pectoris. The absence of pain in these cases is due to the fact that the whole ventricular wall becomes in course of time more or less evenly affected by disease, so that no particular area or areas are unduly stretched during a rise of intra-cardiac pressure. The patient dies from syncope or from rapid dilatation and failure of the heart.

In pseudo-angina the cause of the attack lies in the increased peripheric resistance, and the rise of intra-cardiac pressure produced thereby is evenly distributed over the inner surface of the left ventricle, so that the symptoms are those of acute distension of the heart.

If, now, a portion of the cardiac muscle be insufficiently supplied with blood as a consequence of spasm of one of the coronary arteries or its branches, the conditions for the production of some degree of anginal pain are in all probability present, for the reasons already given.

It is possible, indeed most probable, that spasm of the coronary arteries (which are affected in common with the rest of the arterial system) plays the same rôle in the production of the phenomena of pseudo-angina that organic disease of these vessels does in the causation of an attack of true angina pectoris.

On these lines, therefore, the two forms of angina pectoris merge imperceptibly into one another.

A brief account of the various other theories which have been advanced to explain the phenomena of angina pectoris must now be given.

1. Heberdén ascribed the disease to spasm of the heart.

In view of the morbid changes found in the cardiac walls after death, such a condition of affairs must be a physical impossibility.

2. The disorder has been attributed to acute distension of the heart consequent on the strain to which the organ is exposed in its endeavour to cope with the increased peripheral resistance.

This view has been fortified by taking into account concomitant spasm of the coronary arteries, which, by temporarily restricting the blood supply to the cardiac muscle, adds to the embarrassment of the heart.

There is little doubt that this explanation holds good so far as the phenomena of angina pectoris vasomotoria (Nothnagel) are concerned.

3. Angina pectoris has been regarded as a neuralgia, or even neuritis of the cardiac nerves, by many eminent authorities.

A modification of this view suggests that the disease is a neuralgia superimposed on an organic basis, or that it is a cardiac neuralgia or neurosis in connection with disturbed innervation of the vasomotor mechanism.

4. An old theory revived (first promulgated, according to Osler, by Allan Burns in 1809) attributes the phenomena of the attack to cramp of the cardiac muscle consequent on restricted arterial blood supply. The diminution of the blood supply, which is felt during increased functional activity only, depends on the narrowing of the coronary arteries produced by arterio-sclerosis (atheroma).

The parallel of this condition in the case of the skeletal muscles, is described by Bouley under the title of "intermittent claudication," and is characterized by loss of power and painful cramp-like seizures in the muscles of the limbs and other parts. Here, owing to the partial or complete occlusion of the nutrient vessels by disease, the blood supply to the affected muscles is inadequate for the display of even a moderate degree of functional activity, though it may be sufficient during rest or slight exertion.

The loss of power and pain experienced both in the case of the heart and skeletal muscles is attributable to the same cause, viz. an insufficient supply of arterial blood and the consequent accumulation of waste products in the muscular tissue.

Under physiological conditions, the functional activity of muscular tissue is attended by dilatation of its blood vessels, which not only admits an increased supply of oxygen, but also hastens the removal of waste products. It is this process which is at fault under the circulatory conditions which obtain in angina pectoris, for obviously the thickened and rigid coronary vessels are unable to respond by changes in calibre to the requirements of the cardiac muscle.

SYMPTOMS

Although the symptoms of the two forms of angina pectoris are in all probability similar in pathogenesis, it will be convenient, for purposes of description and of differential diagnosis, to consider them separately.

The symptoms which characterize an attack of true angina pectoris are as follow :—

The patient, usually while engaged in some trivial exertion, or during emotional excitement—less commonly after exposure to cold or after a full meal, but occasionally during perfect rest—is suddenly seized with severe pain in the region of the heart. The pain, which varies in intensity in different cases, is always agonizing, and is described by the patient as tearing, rending, crushing, or stabbing in character. In other instances the sensation is as if the chest were gripped in a vice, or as if the sternum were being forced back on the spine. The pain usually commences in the præcordial region, and radiates backwards to the spine, and upwards to the left side of the neck and left shoulder; thence down the inner side of the left arm as far as the elbow, but rarely beyond this joint. Less commonly the pain extends over both sides of the chest and neck and down both arms, and in cases of exceptional severity it travels downwards over the abdomen to the testes and lower extremities.

The aspect of the patient is expressive of intense suffering and mental distress, and reflects the sense of impending death, which is the dread accompaniment of the pain. A pallid grey hue spreads over the face, the lips become livid in colour, and a cold, clammy sweat breaks out on the forehead.

With the onset of the paroxysm the sufferer generally remains rooted to the spot, not daring to move or even take a deep breath. In other instances he assumes some posture, such as leaning over the back of a chair, kneeling, lying down, etc., which instinct or experience suggests will bring relief.

The breathing is hurried and shallow, and each short inspiration appears to bring little or no relief to the distressing sense of air hunger which is experienced. The patient may feel faint and giddy during the attack, but with very few exceptions consciousness is retained. Flatulence, vomiting, dysphagia, loss of voice, and an overpowering desire to make water occasionally form part of the paroxysm.

PHYSICAL SIGNS

The action of the heart is usually slightly increased in frequency, and it may be irregular. In some cases the action of the heart has been said to be undisturbed. The cardiac impulse is feeble, while the sounds of the heart are, as a rule, short, sharp, and weak. Adventitious sounds in the form of murmurs may or may not be observed.

The pulse is most commonly increased in frequency, irregular, small in volume and force, and ill-sustained, reflecting thus the weak condition of the left ventricular impulse.

In some instances arterial tone is increased, and the pulse gives evidence of heightened tension; that is, the vessel is full between the beats and not easily compressible.

In other cases the pulse shows the condition described by Sir William Broadbent as "virtual tension," that is, a condition which would be high tension if only the heart were strong enough to cope with the increased peripheral resistance.

Cutaneous hyperæsthesia, in the shape of tenderness, can be elicited in the præcordial, postcordial, and supra-orbital regions in cases of angina pectoris.

COURSE AND TERMINATIONS

The duration of the attack varies considerably. It may last for a few seconds, or it may continue for five, ten, or fifteen minutes, and in very rare instances it may extend over hours. As a rule, the duration of the paroxysm is measured by seconds or a few minutes only.

The attack usually ceases as abruptly as it begins. The termination is frequently signaled by the eructation of gas from the stomach, accompanied, it may be, by an urgent desire to micturate. It will be noticed that distension of the stomach by gas is an effect as well as a cause of angina pectoris. The patient is usually more or less exhausted after a paroxysm.

The first attack sometimes ends fatally, but more commonly it terminates in recovery, in which case the patient may remain free from the disease for many years, or possibly for the rest of his life. The seizures tend, however, to increase in frequency and severity, as well as to be more easily excited.

It sometimes happens that the patient is able to ward off or cut short a paroxysm, by immediately discontinuing movement, or by taking drugs the moment the premonitory symptoms of the attack are experienced.

Angina pectoris usually terminates sooner or later in sudden death during a paroxysm. A certain number of the cases develop

syncopal attacks without pain (*angina pectoris sine dolore*), in one of which they die. In some instances gradual failure of the circulation is the cause of death, and according to Balfour this is the usual mode of termination of *angina pectoris*.

In pseudo-angina, or *angina pectoris vasomotoria*, the attack, which is commonly excited by cold or emotion, begins quite suddenly with pain and oppression in the region of the heart, and coldness or numbness of the extremities. The pain may radiate over the chest and upper extremities, but is not so severe as in true angina, and though it may be accompanied by a feeling of anxiety and mental distress, the apprehension of immediate death is not experienced. The sense of air-hunger is a marked feature of the attack, and there is frequently a good deal of restlessness and struggling for breath. The face is pale and the expression anxious, and the extremities are commonly cold and livid.

The action of the heart is excited, and there is usually accentuation of the aortic second sound.

The pulse is frequently irregular in force and frequency, the artery is small and can usually be felt between the beats; the pulse wave, though small, is not easily obliterated by pressure with the fingers.

The attacks are of short duration, seldom lasting longer than a few seconds or a few minutes at the most. The subsidence of the paroxysm is usually marked by the discharge of a large quantity of urine.

The attacks tend to occur in groups, with long periods of complete immunity between them.

DIAGNOSIS

The diagnosis of *angina pectoris* seldom presents much difficulty provided the characteristic features of the attack are developed. The peculiar nature of the pain, its sudden onset and abrupt cessation, its remarkable distribution, together with the terrible sense of impending death, form a group of symptoms that are quite distinctive.

In cases of doubt the various extra-cardiac conditions, which, by giving rise to paroxysmal attacks of pain, may simulate the agony of *angina pectoris*, have to be excluded.

A careful consideration of the symptoms and signs would suffice to distinguish the pain associated with hepatic, renal, or intestinal colic from the pain excited by the cardiac lesion.

The chief difficulty is connected with the differentiation between the organic and functional forms of *angina pectoris*. The main points in the differential diagnosis are as follow :—

True angina is rare before forty-five years of age, apart from

aortic regurgitation, aortitis, and aneurysm. On the other hand, pseudo-angina may occur at any age.

True angina rarely affects women at any period of life. The exciting cause of the attack has considerable diagnostic significance. True angina is most commonly brought about in the first instance by exertion, whereas pseudo-angina comes on spontaneously during rest, or occurs after food, or exposure to cold.

The phenomena of the attack with respect to the severity of the symptoms, as shown by the appearance and behaviour of the patient, are of importance, in that the absence of the facial expression of pain and alarm would be evidence in favour of the incidence of the functional form of the disease.

Moreover, pseudo-angina is commonly associated with other symptoms of nervous disturbance, whereas this is not usually the case with respect to true angina. Evidence of vaso-motor disturbance is more commonly obtained in relation with pseudo-angina, but its presence does not exclude true angina.

It must be borne in mind that, even after the most exhaustive analysis of the symptoms and signs, the diagnosis sometimes remains in doubt.

PROGNOSIS

The attempt to forecast the course and duration of angina pectoris is fraught with many difficulties. Speaking generally, the outlook is favourable in proportion to the extent to which the cause of the paroxysms can be mitigated or removed.

If the attack depends on disease of the cardiac wall alone, the prognosis is bad. On the other hand, if the exciting cause can be shown to be exertion, indigestion, etc., it is possible by the careful avoidance of these conditions to put off the attacks indefinitely. Again, habitually high arterial tone can be lessened by suitable treatment, and in this way the work of the heart can be reduced.

The more easily the attacks are excited the greater the danger.

The prognosis of pseudo-angina is favourable as regards the danger to life, but in view of the occasional difficulty of differentiating between the two forms of angina, it is well to give a guarded prognosis in these cases.

TREATMENT

The objects of treatment in angina pectoris are (1) the relief of the paroxysms, and (2) the removal or mitigation of the conditions upon which the paroxysms depend.

In the treatment of the attacks it is important to discover, if

possible, whether the inability of the heart to perform its work is due to (a) increased peripheric resistance, (b) weakness of the cardiac walls, (c) a combination of the two conditions just mentioned, or (d) interference with the action of the heart by a distended stomach, etc., in conjunction with increased peripheric resistance or cardiac weakness.

If the pulse tension be high during the paroxysm, three or four minims of a one per cent. solution of nitro-glycerine by the mouth, or five to ten minims of amyl nitrite by inhalation, will relax the peripheral vessels, and will thus bring immediate relief to the heart by reducing the stress in front. This plan of treatment is often brilliantly successful in bringing the paroxysm to an end.

When there is no evidence of increased arterial tone and the symptoms point to cardiac weakness only, the best remedy is the subcutaneous injection of three or four minims of liq. strychninæ. Ether, ammonia, or brandy may also be used to stimulate the heart under these circumstances.

A combination of any of these remedies, with a few minims of a one per cent. solution of nitro-glycerine, will meet the requirements of a weak heart acting against increased peripheric resistance.

Distention of the stomach can be removed by the administration of carminatives, such as peppermint or oil of cloves, or by a draught of the compound spirits of ether, or the aromatic spirits of ammonia. In extreme cases the condition can be relieved by the use of the œsophageal tube.

If these methods of treatment fail to do good, opium, given preferably in combination with atropine, either by the mouth or by subcutaneous injection, relieves the pain and gives the heart time to recuperate by rest.

The inhalation of oxygen is sometimes of very great service in relieving the sense of air-hunger which is frequently a prominent feature of the paroxysm.

An attack of angina pectoris vasomotoria, when due to cold, can be relieved by the application of warmth to the extremities in conjunction with vigorous friction of the limbs.

The treatment of pseudo-anginal attacks should be conducted on the lines already indicated in the case of true angina.

After the subsidence of the paroxysm the whole case must be thoroughly investigated with the object of elucidating the cause of the disease.

Errors of diet must of course be corrected, and indigestion treated by appropriate remedies. The food, which should contain a fair proportion of nitrogenous principles, must be light and easily digestible. The quantity of food taken at one meal should be moderately restricted, and a period of rest after eating must be insisted upon.

The use of alcohol, tea, tobacco, etc., must be carefully regulated, and in some instances entirely prohibited.

Exercise in the open air is of the utmost importance, but care must be observed that the amount of exertion undertaken is well within the limits of the cardiac powers. Under no circumstances should exercise be carried to the point of fatigue.

Mental strain and excitement is likewise prejudicial, and the patient should also be warned against exposure to cold and wet. The bowels should be carefully regulated, since constipation is sometimes an exciting cause of the attack.

The chief indication, so far as the therapeutic treatment of angina pectoris is concerned, is the control and reduction of arterial tone.

The methods by which undue resistance in the arterio-capillary circulation can be reduced and kept under control are already familiar to the reader.

Provided the heart exhibits even a moderate degree of vigour, a mild mercurial laxative taken once or twice a week is of the utmost service in reducing arterial blood pressure. The iodide and bicarbonate or citrate of potassium can also be used for the same purpose, and in combination with *nux vomica* or arsenic are the most useful drugs for continuous administration.

Among the direct vaso-dilators the most suitable drug for prolonged use is erythrol tetranitrate, given in half to one grain doses twice daily.

A course of phosphorus is sometimes of great service in the treatment of angina pectoris.

The treatment of aortic disease, when complicated by angina pectoris, should be conducted on general principles.

The medicinal treatment of pseudo-angina consists in the reduction of undue arterial tone and the amelioration of the general health of the patient.

Tonics, in the form of iron, arsenic, etc., are useful in some cases, while in others nerve sedatives, such as the bromides, belladonna, or valerianate of zinc, etc., are indicated.

The hygienic and dietetic measures mentioned in the account of the treatment of true angina are of equal service in the treatment of the functional forms of the disease. In both the organic and functional varieties of the disorder a period of rest must be strictly enforced after the occurrence of a paroxysm.

CHAPTER XXIV

FUNCTIONAL DISORDERS OF THE HEART

Definition—Ætiology—Pathogenesis—Section I. Cardiac Pain ; Local and Referred—Section II. Palpitation ; Definition ; Causation ; Features of Paroxysm—Section III. Cardiac Asthenia—Section IV. Alterations in the Rhythm of the Heart's Action ; Arrhythmia ; Intermittence and Irregularity—Section V. Alterations in the rate of the Heart's Action—Section VI. Tachycardia—Section VII. Decrease in the rate of the Heart's Action—Section VIII. Diagnosis ; Prognosis ; Treatment.

THE so-called "functional diseases" of the heart, with very few, if any, exceptions, are not diseases in the ordinary acceptation of the nosological significance of this term. They are rather the symptoms of disease, or of conditions producing disease, which lead as part of their manifestation to derangement of the nervous and often of the muscular mechanism of the heart.

In other words, "functional disorders" of the heart form part of many different groups of symptoms, which severally constitute "a disease" properly so-called.

A clear grasp of this conception will go far to dissipate much of the difficulty and confusion that has grown up round the subject of functional affections of the heart.

Nevertheless, it has been customary, and it is no doubt convenient, to consider individually a somewhat miscellaneous collection of symptoms, which have this feature in common that they are referable to disturbance, in varying degrees, both of the motor and sensory functions concerned in the regulation and control of the action of the heart.

The adoption of this course of procedure here, in the light of the explanation given above, ought not, therefore, to give rise to any serious misunderstanding.

The chief manifestations of functional disturbance of the heart will be, then, pain, palpitation, and alterations in the rhythm and rate of the organ.

A general survey of the ætiological and pathological conditions which underlie functional disorders of the heart will precede the detailed consideration of the individual symptoms.

ÆTIOLOGY AND PATHOLOGY

The conditions under which disturbance of the nervous and muscular mechanisms concerned in the production and regulation of the beat of the heart may arise are briefly as follow :—

1. Cardio-vascular system.

(a) *Affections of the cardiac nerves and ganglia.*

Pigmentary, fatty, sclerotic, and other changes.

(b) *Affections of the pericardium.*

Pericardial effusion, adherent pericardium.

(c) *Affections of the myocardium.*

Inflammatory lesions, fatty, fibroid, and other changes of a less obvious but not less important kind, consequent on alterations in the nutrition of the muscle fibres.

(d) *Affections of the aorta and coronary vessels.*

Dilatation and aneurism of the aorta. Atheroma of the aorta and coronary arteries.

(e) *Vaso-motor changes in the direction of sudden increase or decrease of arterial tone.*

2. **Nervous system.**—Fright, grief, shock, and depressing emotions of all kinds are prolific sources of cardiac disturbance. Scarcely less important causes comprise nervous exhaustion from overwork, worry, anxiety, want of sleep, etc. Neurasthenia and hysteria.

Again, apoplexy, concussion of the brain, epilepsy, cerebral tumours, etc., are concerned in the production of cardiac irregularity.

Among other causes are lesions of the medulla and spinal cord (locomotor ataxy), mania, melancholia, general paralysis of the insane.

Ex-ophthalmic goitre may for the sake of convenience be included under this head.

3. **Alimentary system.**—Flatulent dyspepsia is a potent cause of functional disturbance of the heart's action. Other conditions are diseases of the stomach and liver, jaundice, constipation, etc.

4. **Respiratory system.**—Emphysema, the early stages of pulmonary tuberculosis and so forth, are sometimes found in association with functional disease of the heart.

5. **Genito-urinary system.**—Inflammatory affections of the kidneys and uræmia. Uterine and ovarian disorders. Masturbation and sexual excesses.

6. **Constitutional and febrile disorders.**—Anæmia, chlorosis, gout, rheumatism, and diabetes. The acute infectious fevers, as for instance, diphtheria, influenza, etc. In convalescence from acute fevers, such as enteric fever, pneumonia, etc.

7. **Toxic influences.**—The abuse of tea, coffee, alcohol, and tobacco. In poisoning by lead, digitalis, belladonna, aconite, etc.

8. **Hygienic, dietetic, and other influences.**—Unhealthy hygienic surroundings, insufficient or unwholesome food, and sedentary occupations are predisposing causes of irregular cardiac action. Over-feeding is no whit less important as a cause of functional disturbance of the heart. Prolonged muscular effort, or occasional bursts of severe exertion, more especially when made in conjunction with mental excitement and insufficient food, are a fertile source of functional disorders of the heart.

Nervous disturbance of the heart's action is more common among women than men, and is particularly apt to arise at the time of puberty, and during the climacteric period.

It will appear, therefore, that disturbance of the neuro-muscular mechanism of the heart may be due to a large variety of conditions, which, however, can operate in three directions only, viz. (1) centrally, (2) locally, and (3) reflexly.

The nervous machinery concerned in the regulation of the beat of the heart is the cardio-inhibitory centre in the medulla, the tenth nerves or vagi which convey "inhibitory" impulses, and the sympathetic nerves which transmit "augmentor" or "accelerating" impulses to the heart.

The local mechanism comprises nervous and muscular parts, through which the rate and rhythm of the cardiac beat may be variously modified.

Finally, in considering the influences which regulate or modify the beat of the heart, it is necessary to include the effect of vaso-motor changes. Stated briefly, the relation of the beat of the heart to blood pressure is that "the rate of the beat is in inverse ratio to the arterial pressure" (Foster). In other words, a rise of blood pressure is accompanied by a decrease, while a fall of blood pressure is attended by an increase in the rate of the heart beat. This circumstance partly accounts, no doubt, for the acceleration of the heart in fevers and other conditions.

In health the rate and rhythm of the heart vary within wide limits under the influence of the fluctuations of ordinary blood pressure, which through the administration of the central nervous system is regulated in accordance with the ever-varying needs of the organism.

Normally the action of the heart is in all probability kept under continuous control by impulses passing along the vagi from the cardio-inhibitory centre. Acceleration of the heart, therefore, may be due either to relative or absolute loss of vagus control. Thus stimulation of the sympathetic nerves may give rise to impulses which overpower the normal action of the cardio-inhibitory mechanism, or the function of the vagi may be more or less in abeyance.

On the other hand, stimulation of the vagi either locally or through the cardio-inhibitory centre will lead to slowing of the heart.

Furthermore, since in disease loss of functional activity proceeds *pari passu* with the degree of its specialization, the controlling machinery of the heart will fail before the accelerating mechanism, and herein may lie part of the explanation of the rapid cardiac action in cases of fever, poisoning, and so forth.

Alterations in the rate and rhythm of the heart may also depend on interference with the nutrition of the organ, or on the direct action on the muscular substance of imperfectly oxidized products of metabolism, or of poisons circulating in the blood (cf. irritable heart, fatty heart, gout, rheumatism, jaundice, etc.).

Again, local or reflex excitation of the cardio-inhibitory, vaso-motor, and cardio-augmentor (?) centres must be a prolific source of disturbance of the cardiac action.

Under normal conditions the harmonious co-operation of these centres, under the influence of local and reflex stimuli, regulates the blood pressure, and hence the blood flow in accordance with the demands of the system by alterations in the rate and rhythm of the heart. It is not surprising, therefore, that under conditions of disease this complex mechanism becomes deranged with corresponding modifications in the rate and rhythm of the heart.

Palpitation is an instance of such abnormal action. Here, as the result of emotion or other mental disturbance, the action of the cardio-inhibitory centre itself is suddenly inhibited, or there is a sudden increase of the augmenting impulses along the sympathetic nerves which overpowers the normal controlling mechanism of the heart, or possibly there is a combination of these conditions. Nor does this exhaust the possible methods by which the attack may be explained, for emotional disturbance leads also to sudden vaso-motor changes, either in the direction of arterial constriction or relaxation, and in this way may give rise to the phenomena of the paroxysm.

Take again the case of fevers, which by producing alterations in the quantity and quality of the blood give rise not only to a fall of blood pressure and stimulation of the cardiac accelerating mechanism, but also to more or less direct poisoning of the muscular substance of the heart.

The heart, therefore, with respect to the neuro-muscular machinery

concerned in the regulation of its beat, may be attacked either centrally, locally, or reflexly, as well as through the collateral channel of the vaso-motor mechanism.

It will thus appear how complex may be the operation of the conditions which give rise to alterations in the rate and rhythm of the heart.

The mode of origin of the pain and other distressing sensations which may accompany the symptoms comprised under the term "functional disorders of the heart" is not fully understood.

Normally the cardiac afferent impulses which travel along the vagi and sympathetic nerves do not give rise to any perceptible impressions; in other words, we are ordinarily unconscious of the action of the heart.

Now, an increase in the intensity of the peripheral stimuli, and hence of the cardiac afferent impulses, or an irritable condition of the nervous structures engaged in transmitting and receiving these impulses, might give rise to sensation of a more or less painful nature.

Thus in organic disease of the heart, whether valvular or mural, the increase of intra-cardiac pressure, and the accompanying distension of one or other of the chambers of the organ, must give rise to undue stretching and compression of the peripheral cardiac nerves, and in this way to irritation of these structures which may be felt as pain. Again, in conditions of nervous irritability, which must also involve the cardiac innervation (both central and peripheral), the ordinary afferent impulses may be sufficient to give rise to painful impressions. Furthermore, if an irritable condition of the cardiac nerves (both central and peripheral) be associated with an increase in the intensity of the peripheral stimuli *à fortiori*, a still greater effect would be produced, and the painful impressions may then extend and overflow to other nerves in connection with the cardiac innervation, as in the case of angina pectoris, and other organic affections of the heart.

The pain, therefore, that is occasioned by diseases of the heart may be either local or referred.

According to Head, local pain is limited strictly to the præcordial area, and is not accompanied by superficial tenderness. It is, however, usually attended by deep tenderness, which is increased by pressure in direct ratio to the force applied. Further, local pain is not accompanied by that form of headache which is associated with tenderness of the scalp.

Referred pain, on the other hand, is distributed over more or less well-defined areas situated both on the anterior and posterior aspects of the chest wall, and also in other parts of the body, and is accompanied by superficial tenderness, which is usually relieved by pressure.

Referred pain is likewise commonly attended by headache and superficial tenderness of the scalp in the supra-orbital and temporal

regions. For further information on this subject the reader is referred to Dr. Head's paper in *Brain* (lxxiv. and lxxv.).

The following account of the symptoms of functional diseases of the heart do not apply to the similar manifestations associated with organic lesions of the organ, for the reason that these have already been considered in other sections of this work.

SECTION I

CARDIAC PAIN

Local cardiac pain is probably more often complained of in connection with functional than with organic disease of the heart.

The pain is most commonly felt over the region of the apex beat, less frequently in the third or fourth left intercostal space; but it may be experienced over any portion of the præcordial region. It is usually possible to evoke a greater or less degree of tenderness by pressure over the seat of pain. Care must, however, be taken not to confound true local pain with referred pain, which is also frequently experienced over the præcordial region in cases of functional disease of the heart.

Cardialgia is apt to occur in paroxysms, and is very commonly associated with breathlessness, or oppression of breathing. The pain is variously described; at one time it is tearing, cutting, stabbing, or burning in character; at another it is a dull, heavy, persistent ache. The suffering of the patient is often very greatly intensified by the fear that he or (more commonly) she is the subject of heart disease.

The pain is generally increased by fatigue or emotional disturbance, and sometimes by exertion; on the other hand, moderate exercise may bring relief to the pain. Attacks of palpitation, when they occur, usually aggravate the suffering of the patient.

Local cardiac pain may arise in various ways. Thus it may depend on intrinsic causes, such as have already been considered. It may also be due to extrinsic conditions, as, for instance, the direct pressure exerted on the heart by distension of the stomach or abdomen, or by fluid or air in the pleural cavity.

Again, reflex disturbance of the heart, often associated with pain in the præcordial area, is very commonly produced by sudden emotion, or by prolonged grief or anxiety.

The pain over the heart that is so frequently found in connection with conditions of nervous strain or exhaustion, digestive derangements, and uterine or ovarian disorders is also due to reflex disturbance of the organ.

SECTION II

PALPITATION

Palpitation is essentially a paroxysmal affection characterized by frequent, forcible, and oftentimes irregular action of the heart, which is perceptible to the sufferer.

A variety of the disorder, sometimes termed false palpitation, is distinguished by a distressing subjective sensation of cardiac action, although on physical examination no appreciable alteration in the rate or rhythm of the heart can be detected. On the other hand, the most extreme derangement of the heart's action, both as regards rate and rhythm, may be found without any consciousness of the disturbance.

The distinctive feature of palpitation is, therefore, a more or less distressing perception of the beating of the heart.

The predisposing causes of palpitation, apart from organic disease of the heart, are loss of tone or undue excitability of the nervous system, neurasthenia, hysteria, mental or physical exhaustion, general loss of tone, as, for instance, is observed after the acute fevers and other exhausting diseases, masturbation and sexual excesses, and certain disordered conditions of the blood, to wit, lithæmia, anæmia, and the like. Palpitation is more common among women than men, and is especially apt to arise during periods of nervous stress and excitement, such, for instance, as are observed at the time of puberty and the climacteric.

Among the chief exciting causes of palpitation may be mentioned sudden emotion, shock, grief, fright, etc., violent exercise, digestive disturbances, especially when attended by flatulence, uterine and ovarian disorders.

The abuse of alcohol, tobacco, tea, coffee, etc., is also a common exciting cause of palpitation.

A brief reference must be made here to the condition described by Da Costa under the title of *irritable heart*.

According to Allbutt it is possible to distinguish two classes of this disorder; the one, a curable affection occurring for the most part in young men; the other, frequently an incurable condition, observed principally among soldiers (hence called by Allbutt "the soldier's heart").

The irritable heart of young people is found chiefly in ill-developed, nervous, and dyspeptic subjects, and is the product of occasional bursts of more or less severe muscular exertion in conjunction with irregular habits with respect to the taking of food and the use of alcohol, tea, coffee, tobacco, and the like.

The symptoms of this form of irritable heart are palpitation

and more or less constant præcordial uneasiness or pain with easily excited dyspnœa. Physical examination of the chest reveals excited action of the heart, sometimes in association with a little dilatation of the organ, and occasionally with slight hypertrophy.

The blood pressure in these cases is nearly always low.

"The soldier's heart" is usually observed among raw recruits, and is due to cardiac overstrain consequent on the severe muscular exertion entailed by drilling, forced marches, etc., in conjunction with mental excitement and insufficient training, or with an enfeebled state of the muscular tissues following attacks of fever or diarrhœa, insufficient food, and the abuse of alcohol and tobacco.

The symptoms of this variety of irritable heart are palpitation, cardiac pain, dyspnœa on exertion, and a tendency to the production of a great increase in the pulse rate on very slight provocation.

The heart is usually somewhat enlarged, more in the direction of dilatation than of hypertrophy, while the cardiac sounds show but little deviation from the normal.

The pulse rate is increased, and the blood pressure is low.

This condition of irritable heart is of importance from the fact that it eventually gives rise to hypertrophy of the organ, and possibly, in some instances, to valvular disease. Dilatation of the heart ultimately supervenes with its effects.

The ordinary forms of neurotic palpitation seldom, if ever, give rise to organic disease of the heart.

An attack of palpitation usually commences abruptly, sometimes with, but perhaps more commonly without, an obvious exciting cause. It not infrequently happens that the patient is waked up from sleep by the tumultuous beating of the heart. The excited and violent pulsation of the organ is often attended by more or less præcordial uneasiness or discomfort, seldom amounting to actual pain. A distressing feeling of choking or suffocation, accompanied by oppression of the breathing, is frequently experienced. Violent throbbing of the arteries, noises in the ears, giddiness, faintness, and a sense of fulness in the head are not uncommon features of the paroxysm.

The face may be pale or flushed, and bedewed with sweat, while the features are frequently expressive of anxiety or alarm.

The attack may last a few minutes, or may extend over several hours. It usually ceases abruptly with a kind of shock, but in some instances a more gradual subsidence of the symptoms is observed. The termination of the paroxysm is sometimes marked by the passage of a large quantity of pale-coloured urine. An attack of palpitation is followed by a greater or less degree of exhaustion.

An examination of the heart, during a paroxysm, usually shows an increase in the area of visible cardiac pulsation.

Palpation may discover forcible action of the heart, but more

often the impulse of the organ is felt as a slap or tap, and in cases of extremely rapid cardiac action, as a mere vibration only.

The area of cardiac dulness is not increased.

The sounds at the apex are usually short and loud, while at the base the second sound may be accentuated, but more commonly it is weak. ¹ Approximation of the sounds is the rule, but when the heart is acting very rapidly, the two sounds become equidistant in their time relations, and more or less similar in character, so that the foetal heart beat is very closely simulated.

Murmurs are not infrequently audible during an attack of palpitation. They are systolic in time, and are heard most commonly at the apex of the heart. The method of production of these temporary murmurs is uncertain; while some, no doubt, are of hæmic, or cardio-pulmonary origin, others are, in all probability, due to relative incompetence of the mitral valve.

The pulse reflects the excited condition of the heart.

Irregularity of the pulse is, in some instances, due to the failure of the heart to transmit the pulse wave as far as the radial artery. The tension of the pulse may be high or low, under the influence of the vaso-motor changes which are so commonly found in association with attacks of palpitation.

SECTION III

CARDIAC ASTHENIA

Under the title of "cardiac asthenia," Da Costa has comparatively recently given an account of a functional disorder of the heart which he distinguishes from irritable heart, and which, in all probability, has, up to the present, been included under the somewhat indefinite description of "weak heart." A patient of the author's, who probably suffered from this variety of functional disorder, complained of a "tired heart," a description which more or less exactly fits the affection in question.

The malady may occur at almost any age, in either sex, and is especially apt to attack persons who have been exposed to nervous strain.

The incidence of the disorder is always sudden, and is accompanied by a greater or less degree of prostration, manifested by muscular feebleness, frequent sighing and yawning, and a tendency to faint on the least exertion.

There is no shortness of breath, or other disturbance of respiration. A general feeling of chilliness may be experienced, and the temperature is subnormal.

Sleeplessness is not an uncommon feature of the disorder.

A sense of præcordial discomfort more distressing than actual

pain is experienced, and the patient is hardly ever without a sort of semi-consciousness of the heart's action.

Examination of the heart demonstrates a feeble, possibly slightly diffused impulse. The percussion dulness is normal. At the apex the sounds of the heart are short and feeble; the second sound at the base is not accentuated.

In some instances the action of the heart is irregular.

Murmurs, more especially at the apex, may be present.

The pulse is small, and of extremely low tension.

There are no signs of circulatory disturbance beyond those already mentioned.

The disorder generally persists for some months, and recovery, though usually complete, is very slow.

SECTION IV

ALTERATIONS IN THE RHYTHM OF THE HEART'S ACTION

ARRHYTHMIA

The meaning of the word "rhythm" as applied to the action of the heart is the numerical harmony or proportion of the beats of the organ.

Alterations in the rhythm of the heart's action may take place in two directions, *i.e.* (1) by the occasional omission of a beat, and (2) by the appearance of beats at unequal intervals of time, which practically necessitates inequalities in the force and volume of the beats.

These deviations from the normal rhythm are known respectively as "intermittence" and "irregularity" of the heart's action.

Either abnormality may occur independently, but in a large number of cases they are combined. Regular irregularity of the cardiac rhythm is sometimes described under the term "allorhythmia."

Intermittence of the heart's action may occur at regular or irregular intervals. Intermittence of the pulse does not necessarily mean the dropping of a cardiac beat, since the ventricular contraction, though audible, may not be sufficiently powerful to propel the pulse wave as far as the radial artery.

Intermittence of the heart's action is for the most part a sign of trivial significance. It is, however, sometimes found in association with fatty degeneration of the cardiac wall, and may then, if habitual, be of grave import. Its occurrence, too, in connection with cerebral disorders and acute affections of the lungs is also of serious augury.

Irregularity of the heart's action may be incidental or more or less constant. It is occasionally of little or no importance, but it is, generally speaking, always of much graver significance than intermittence, more especially in connection with organic disease of the heart. A combination of intermittence and irregularity of the cardiac action under these conditions increases the gravity of the prognosis. As a rule irregularity of the heart's action is more serious when habitual than when occasional.

The following disturbances of the cardiac rhythm are observed in addition to those already mentioned, as the result of the operation of one or both of the factors concerned in the production of arrhythmia.

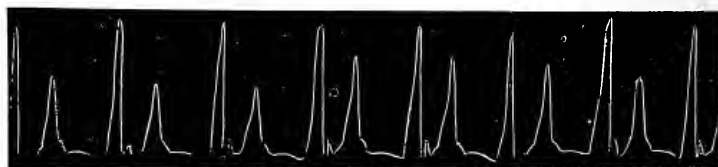


FIG. 42. BIGEMINAL PULSE TRACING

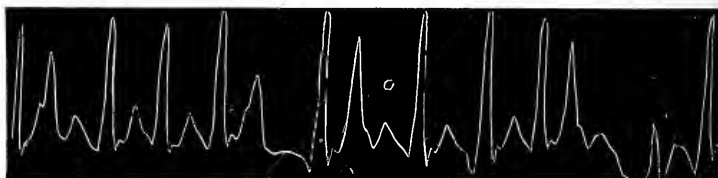


FIG. 43. TRIGEMINAL PULSE TRACING

The alternate heart beat corresponding with the *pulsus alternans* is produced by repeated and successive strong and weak ventricular contractions.

A duplicate or triplicate arrangement of the cardiac action may be observed, so that groups of two or three beats occur with a pause between each group.

The *pulsus bigeminus* and *pulsus trigeminus* are respectively the peripheral expression of these two forms of cardiac arrhythmia. In the *pulsus bigeminus* the second beat is usually the weaker of the two. The weakness of the second beat may be carried to the point of extinction, so that two beats of the heart occur to one of the pulse.

These varieties of arrhythmia occur most commonly in mitral stenosis, more especially when under the influence of digitalis.

The alteration in the rhythm of the heart's action which gives rise to the *pulsus paradoxus* is an illustration of the influence of respiration on a weak or hampered left ventricle.

The most extreme disturbance of the cardiac rhythm is observed in the condition known as *delirium cordis*.

The alterations in the rhythm of the heart's action associated with approximation, spacing and reduplication of the cardiac sounds, together with their significance, have been considered in previous sections (see pp. 50 and 51).

The chief causes of disturbance of the cardiac rhythm, exclusive of structural lesions of the heart, are functional and occasionally organic affections of the brain, the acute specific fevers, and acute disease of all kinds, but more especially of the lungs and kidneys, flatulent dyspepsia, and reflex irritation from gastro-intestinal and hepatic derangements, sexual irregularities, and the like.

Arrhythmia is also commonly due to the abuse of tobacco, tea, alcohol, etc., and to the action of certain drugs, notably of digitalis, belladonna, and aconite.

Gout and allied disorders are also productive of intermittent and irregular action of the heart.

It frequently happens that cardiac intermittence or irregularity lie outside the sphere of consciousness, and this is especially likely to be the case when the disturbance of rhythm is associated with organic disease of the heart, and is therefore more or less constant. On the other hand, intermittent action of the heart is not uncommonly attended by a fluttering or sinking sensation in the præcordial area, followed by a thump or bump as the cardiac action is resumed with a somewhat more powerful contraction than usual.

Apart from palpitation, irregular action of the heart is rarely perceptible to the patient. It is said that disturbance of the cardiac rhythm is of less moment when attended by subjective sensations than when it is not.

Physical examination of the heart in cases of cardiac intermittence sometimes shows that the dropping of the beat is apparent rather than real, since there is evidence that the ventricular contraction occurs but is not sufficiently powerful to transmit the pulse wave as far as the wrist.

In some instances, however, a real omission of the beat takes place, so far as the absence of auscultatory and other physical signs can demonstrate this occurrence.

SECTION V

ALTERATIONS IN THE RATE OF THE HEART'S ACTION

Alterations in the rate of the heart's action lead either to an increase or to a decrease of its frequency, and these are the aspects under which the subject will be considered.

1. Increase in the Rate of the Heart's Action

Increased frequency of the heart's action, which may amount to eighty, ninety, or even one hundred beats per minute, is a normal phenomenon in some individuals. Otherwise the causes of an increase in the rate of the heart's action are practically any of the conditions that were enumerated under the heading of "ætiology" at the beginning of this chapter. Palpitation and rapid heart rate are very commonly associated, but they may and frequently do occur independently.

An increase in the frequency of the heart's action is symptomatic of the affection known as ex-ophthalmic goitre, and it is found sooner or later in almost all forms of organic disease of the heart. It is also observed in connection with certain lesions of the vagus nerve and medulla oblongata.

The effect of an increase in the rate of the heart's action on the time relations of the various events comprising the cardiac cycle is to shorten the diastole. The shortening of the diastole interferes with the proper filling of the heart with blood, so that not only is the output per ventricular contraction greatly reduced, but also the gross output per unit of time is not increased, and may be diminished.

It is obvious, therefore, that rapid action of the heart does not *per se* add to the work of the organ, and herein may lie to a large extent the explanation of the absence of cardiac hypertrophy, even after long-continued increase in the rate of the heart's action.

SECTION VI

TACHYCARDIA

Paroxysmal attacks of extremely rapid cardiac action, attended by palpitation and more or less præcordial discomfort, are distinguished under the title of tachycardia, which may possibly represent a true neurosis of the heart.

Tachycardia is found chiefly among neurotic subjects of either sex, and it may occur at almost any age. In some cases among women the attacks have appeared for the first time about the period

of the menopause. An hereditary influence has been remarked in occasional instances.

The causation of tachycardia has not yet been satisfactorily determined. The attacks, in some instances, have appeared to depend on emotional disturbance, gastric derangements, uterine displacements, pregnancy, floating kidney, over-exertion, and so forth. In other cases the abuse of tobacco, alcohol, etc., has appeared to exert a causal influence in the production of the disorder. In a case observed by the author, the attacks occurred apparently as a sequel of acute rheumatism.

The pathogenesis of tachycardia is equally uncertain.

The rapid action of the heart has been explained on the supposition of loss of vagus control, or of discharging lesions affecting the "accelerating" centres, or of a combination of these causes. Lesions of the cardiac ganglia and vaso-motor changes have also been assigned a share in the causation of the attacks.

As a working hypothesis it may be supposed that in cases of tachycardia there is an unstable condition of the neuro-muscular mechanism concerned in the regulation of the heart's action, which would affect chiefly the controlling function of the vagi and cardio-inhibitory centre as well as of the vaso-motor centre. Under these circumstances reflex or centric irritation of the neuro-muscular apparatus of the heart might, through the accelerating mechanism, be capable of giving rise to the phenomena of the attacks.

The paroxysms commence suddenly with a feeling of tightness, uneasiness, or oppression in the præcordial region, accompanied by a more or less distressing sensation of rapid cardiac action. The degree of discomfort experienced varies greatly in different cases. The pulse rate rapidly runs up to 150, 200, or even 250 beats a minute. In one of the reported cases a pulse rate of over 300 beats a minute was counted (Bristowe).

The heart acts regularly, as a rule, throughout the attack, unless dilatation supervenes, but the pulse may become irregular owing to the failure of some of the beats to reach the wrist.

The duration of the paroxysm varies greatly. It may continue for a few hours or a few days, and, it is stated, that in exceptional instances it has persisted for weeks.

As a rule the attack ceases abruptly, and is followed by more or less exhaustion. A copious secretion of urine may accompany the terminal stages of the paroxysm.

The attacks may recur at intervals of a few hours, days, or weeks, or, as is not infrequently the case, the period of immunity extends into months or even years.

A physical examination of the heart during the attack usually reveals nothing abnormal beyond the modification in the rhythm of the sounds that is always associated with rapid cardiac action. The shortening of the diastole gives rise to the so-called foetal heart rhythm, or tick-tack action of the heart.

Recovery is usually complete, provided the interval between the attacks is sufficiently long to enable the heart to recuperate. In some instances the repeated occurrence of the attacks at short intervals exhausts the heart, and dilatation then supervenes together with its effects. Death, under these circumstances, may be due to gradual failure of the heart or, as is more often the case, to syncope.

SECTION VII

DECREASE IN THE RATE OF THE HEART'S ACTION

The term "bradycardia" or brachycardia is used to denote slow action of the heart. It has no other significance.

An infrequent action of the heart, by which is meant a rate below fifty beats a minute, is a normal condition in some individuals.

A decrease in the rate of the heart's action is a physiological occurrence in parturition (Osler). It is also found in association with hunger.

A slow action of the heart is sometimes observed in connection with fatty degeneration, less often with fibroid disease of the cardiac walls. The significance of the phenomenon in this respect is increased when it occurs in association with vertigo and syncopal attacks (Allbutt).

A slow pulse does not necessarily imply an infrequent action of the heart, since some of the beats may fail to reach the periphery. Deductions drawn from the pulse rate with respect to slowing of the cardiac action should be checked, therefore, by an examination of the heart.

Apart from the conditions mentioned above, a decrease in the rate of the heart's action is observed in association with—

1. A rise of blood pressure.
2. Pain.
3. Diseases of the nervous system.

Apoplexy, and tumours of the brain. Lesions of the medulla.

Diseases of the spinal cord (*tabes dorsalis*).

Emotion. Hysteria, melancholia, mania (occasionally).

4. Reflex irritation from gastro-intestinal derangement, worms in the intestinal tract, ovarian and uterine disorders, etc.
5. Exhaustion during convalescence from fever or from over-fatigue, sexual excesses, etc.
6. Toxic influences, such as by poisons associated with the acute specific fevers, diphtheria, influenza, etc., or with lithæmia uræmia, jaundice, and so forth.

The action of lead, tobacco, digitalis, and the like.

7. Stokes Adams' disease.

Slowing of the heart's action is for the most part effected by direct or reflex stimulation of the central or peripheral connections of the vagi. There is, however, both physiological and clinical evidence to show that a like result may possibly depend, to some extent at least, on direct stimulation of the cardiac muscle.

The functions of the cardiac ganglia are not thoroughly understood, but so far as is known these structures exert no influence on the regulation of the heart's beat. Theoretically, diminished functional activity of the cardiac accelerating mechanism might be attended by a reduction in the rate of the heart's action, but the physiological and clinical evidence bearing on this point is not sufficiently convincing to admit of a definite statement.

Syncope or fainting is the only symptom that will be considered in connection with slowing of the heart's action. This phenomenon, which is within the experience of most people at some period of their lives, is most commonly observed in association with pain or emotional disturbance. As there are all degrees of syncope between death and so-called fainting attacks, so are there, no doubt, all degrees of infrequent cardiac action between complete arrest and the reduction in rate necessary to produce symptoms of cerebral anæmia, which is the proximate cause of the phenomena.

In an ordinary fainting attack, due to reflex stimulation of the vagi by pain, emotion, etc., there is probably a reduction in the rate of the heart's action in association with ventricular contractions of so feeble a kind that they are not preceptible within the limits of a physical examination of the heart.

There is an additional factor, frequently a potent one, in the production of fainting attacks of functional origin, viz. vaso-motor disturbance, which, by relaxing the vessels in the cutaneous and splanchnic areas gives rise to a fall of systemic blood pressure.

STOKES ADAMS' DISEASE.

This remarkable symptom-group or syndrome is characterised by the occurrence of bradycardia in association with symptoms that are referable to disturbances of the cardio-vascular, nervous and respiratory symptoms. The ætiology and pathology of the Stokes Adams' syndrome is still obscure, but the conditions under which it may be observed are arranged by Osler in three categories, viz. :—

1. *Post-febrile group*.—In this class the attacks follow, rarely accompany, an acute infection such as enteric fever, scarlet fever, pneumonia, diphtheria, influenza, etc.

2. *Neurotic group*.—In this group the syndrome is associated with a gross lesion of the medulla or some other portion of the central or peripheral nervous apparatus, or with functional disturbance of the nervous system.

3. *Arterio-sclerotic group*.—In this variety there are found structural changes in the heart or vascular system, or in both. The large majority of the cases belong to this group, and they are observed among men over the age of fifty.

The central feature of the Stokes Adams' syndrome, in its clinical aspect is bradycardia, which may be observed in an acute, chronic, or intermittent form in different cases. The slowing of the heart is associated with vertigo, syncopal attacks, epileptiform seizures, pseudo-apoplexy, and respiratory disorders, which will now be briefly considered.

The bradycardia or slowing of the heart beats, as reflected by the pulse, may be real or apparent only, inasmuch as it not infrequently happens that some of the cardiac systoles are abortive, and fail to transmit the pulse wave as far as the radial arteries, or even in some instances as far as the carotid trunks.

A temporary arrest of the heart's action, and vaso-motor disturbances in the form of pallor, sweating, etc., are sometimes observed. Vertigo is a very constant symptom, and may precede by months or years the full development of the syndrome. Syncope with loss of consciousness is usually a prominent feature of the disease. The attacks of fainting come on suddenly, and seldom last more than a minute or two, and are apt to be associated with convulsive seizures of an epileptic type.

The pseudo-apoplectic phenomena resemble the ordinary features of an apoplectic stroke, except that after lasting a few minutes, consciousness is restored and the patient is able to resume his usual avocations.

The respiratory disturbances take the form of asthmatic seizures and Cheyne Stokes' breathing.

The diagnosis of Stokes Adams' disease when fully developed presents little or no difficulty, but in its initial or slighter manifestations the syndrome is very easily overlooked. The occurrence of bradycardia in association with vertigo should in all cases suggest a careful examination of the patient in view of the possible existence of the symptom group under consideration. The prognosis is very grave, and in the present state of our knowledge of these cases treatment can be symptomatic only.

SECTION VIII

DIAGNOSIS

The diagnosis of functional disorders of the heart rests on the presence, either alone or in combination, of the symptoms and signs which have been described. In practice the chief difficulty lies in the differential diagnosis of the cause of the cardiac disturbance,

more particularly with regard to the exclusion of organic disease of the heart.

In purely functional affections of the heart there is an obvious disproportion between the intensity of the subjective symptoms and the degree of mechanical disturbance of the circulation. Moreover, palpitation and arrhythmia of functional origin are sometimes relieved by exertion, which exaggerates these symptoms when found in association with organic disease of the heart. This statement does not of course apply to the palpitation observed in connection with anæmia, but in this condition the cause of the cardiac disturbance is usually obvious.

Physical examination of the heart in cases of functional disorder rarely demonstrates the presence of any serious degree of cardiac enlargement, though it is not uncommon to find a little dilatation of one or both ventricles. Moreover, the character of the sounds in functional cardiac affections is sometimes of service in excluding organic disease of the heart.

Again, murmurs heard in association with functional disorders are practically always systolic in time, and they do not replace the sounds of the heart as in the case of bruits due to organic disease of the organ. Furthermore, the site of greatest audibility and the area of distribution of functional and organic apical systolic murmurs do not coincide.

Apart from organic disease of the heart, the differential diagnosis of the causes of functional affections of the organ rests on the history of the case, on the character of the symptoms, and on the causal indications furnished by the cardio-vascular and other systems.

Tachycardia is distinguished from rapid action of the heart due to other causes by its paroxysmal character. An increase in the rate of the heart's action not uncommonly precedes the development of the proptosis and thyroid enlargement in Grave's disease, but in this disorder the *persistent* acceleration of the pulse would suffice to exclude tachycardia.

Again, dilatation of the heart, mitral stenosis in its terminal stages, and bulbar paralysis also give rise to increased frequency of the pulse; but in these conditions both the rate and rhythm of the cardiac action are more or less persistently altered, features which are inconsistent with tachycardia. A paroxysmal increase in the rate of the heart's action is sometimes observed in connection with the crises of locomotor ataxia. A correct diagnosis seldom presents much difficulty in these cases, inasmuch as the attacks are associated with disturbance of the cardiac rhythm and with the symptoms peculiar to the nervous disorder.

PROGNOSIS

The prognosis of functional disorders of the heart is, on the whole, favourable as regards the expectancy of life. The prospects of recovery, so far as the cure or prevention of the cardiac disturbance is concerned, depends, for the most part, on the extent to which the cause can be removed. In the absence of a discoverable cause, and in view of the difficulty which is so often experienced in excluding organic disease of the cardiac walls, it is advisable, before giving a favourable prognosis in a case of functional disorder of the heart, to make repeated and careful examinations of the patient both in the upright and supine positions.

The outlook with respect to that variety of irritable heart known as "soldier's heart" (Allbutt) is not good, inasmuch as the cardiac disturbance leads ultimately to dilatation and hypertrophy of the heart.

The prognosis of tachycardia depends largely on the duration of the attacks and the frequency of their incidence (Herringham). Long duration (*i.e.* more than four or five days) and frequent repetition (*i.e.* recurrence within a few months) of the paroxysms involve more or less cardiac strain, which sooner or later leads to dilatation of the heart and its disastrous effects. On the other hand, short attacks with protracted periods of immunity are compatible with longevity. The prognosis of bradycardia is the prognosis of the cause of the cardiac disturbance.

TREATMENT

The treatment of functional disorders of the heart consists as much in the general management of the patient as in the employment of drugs. It is obvious that in practice treatment should be directed, in the first instance, to the mitigation or relief of the paroxysms, and, secondly, to the prevention or removal of both the immediate and remote causes of the cardiac disturbance. Nevertheless, for descriptive purposes it is convenient to take the second object first, inasmuch as certain general hygienic, dietetic, and moral considerations are applicable in the treatment of all forms of functional disease of the heart, whereas special means are requisite for the relief of the individual symptoms.

It is of the utmost importance in all cases of functional cardiac disorder to be able to convince the patient that there is no disease of the heart. While, on the one hand, a careful investigation of the condition of the heart should be made at the outset; on the other, too much attention should not be directed to the organ by subsequent examinations. Moreover, the patient's thoughts

should so far as possible be diverted from his ailment by change of air and scenery, by cheerful companionship, by the pursuit of some wholesome hobby, and by the avoidance of strain and excitement of all kinds.

A moderate amount of exercise in the open air is nearly always beneficial, and any outdoor pursuit which does not involve excitement or fatigue may be safely encouraged. Cold bathing, more especially in the form of shower baths, is frequently of service, but should be used with caution. Sea bathing is sometimes attended by the happiest results.

The adoption of an open-air life, and the cultivation of regular habits with respect to the taking of food, alcohol, sleep, and exercise, is often sufficient to effect a cure.

Moderation with regard to the quantity of food, and a common-sense discrimination with respect to its quality are of the first importance. It is frequently advisable to altogether interdict the use of tobacco, alcohol, tea, and coffee.

Sexual excesses are a prolific source of functional disorders of the heart; hence it may become necessary to warn the patient against this form of over-indulgence.

Inasmuch as functional affections of the heart are so frequently found in association with disturbance of the nervous system, an attempt should be made to remove such causes of mental strain as overwork, worry, anxiety, and so forth. Hysteria and conditions of general nervous irritability or excitement may be met by the use of bromides, valerian, hyoscyamus, and other sedatives.

Gastro-intestinal derangements, which are perhaps the most common cause of functional disturbance of the heart, should be corrected by careful dieting, and the exhibition of the remedies appropriate to the relief of the particular kind of dyspepsia that obtains. In children the possible presence of intestinal worms should not be overlooked. The bowels must be regulated, and any source of intestinal irritation, as, for instance, piles, should receive attention. The correction of uterine displacements is sometimes followed by the disappearance of the cardiac symptoms.

Anæmia, gout or lithæmia, and other disordered conditions of the blood, should receive appropriate treatment.

If a careful and systematic investigation of the case fails to reveal the cause of the cardiac disturbance, the drugs most likely to be of service are the bromides, salicylates, or iodides, with or without arsenic, strychnine, or digitalis.

The treatment of cardiac pain depends for the most part on its cause. Digestive disorders, uterine derangements, emotional disturbance, and increased arterio-capillary resistance must be controlled and removed by appropriate means.

The application of a belladonna plaster, or of a small blister, or, if the pain be very severe, of one or two leeches over the painful area, usually gives great relief. Belladonna may also be

given internally with advantage. In the absence of a discoverable cause, or when the pain resists other remedies, the administration of one-sixth of a grain of barium chloride in pill form three times a day sometimes gives excellent results.

An attack of palpitation can sometimes be relieved by exercise or by the simple expedient of drawing a few deep breaths.

In general, the treatment of the paroxysm consists in rest in the recumbent position and in the use of stimulants, such as ether, ammonia, valerian, ginger peppermint, camphor, and the like. The compound spirits of ether, in drachm doses, is an efficient remedy under these circumstances. The following prescription is an example of a very serviceable combination in the treatment of palpitation:—

R Tincturæ Valerianæ Ammoniatæ	ʒi.
Tincturæ Lavandulæ Compositæ	ʒss.
Spiritus Vini Rectificati	ʒi.
Aquæ Chloroformi	q. s. ad. ʒss.
Fiat Mistura.	
S. One tablespoonful to be taken every two or three hours.	

Alcohol, in some warm vehicle, is also of service, but its administration requires strict supervision. The vagi may also be stimulated through the nasal mucous membrane by means of smelling-salts, snuff, ammonia, etc. Hot or cold applications to the præcordial region usually afford relief, but cold, in the form of an ice-bag, must be employed with caution, more especially if the action of the heart be irregular. In the case of less acute and more protracted attacks of palpitation, the various measures just mentioned must be supplemented by treatment directed to the more remote causes of the cardiac disturbance.

With this object the hygienic and dietetic regulations which were detailed at the beginning of this section should be put into operation. The exhibition of nerve sedatives, more especially of the bromide of sodium or ammonium, is usually of service in neurotic cases. Belladonna, given internally, or applied in the form of a plaster to the præcordial region, is sometimes of service. The use of aconite is strongly recommended by Allbutt, but care must be observed in the employment of this remedy. Digitalis is seldom of much benefit, but the administration of *cactus grandiflorus*, as a tincture, is not infrequently attended by excellent results. Vaso-motor disturbances in connection with palpitation should be corrected and controlled by appropriate treatment. Tonics are frequently of service in the intervals between the attacks.

The treatment of that form of irritable heart which occurs in young people consists in the careful regulation of the habits of the patient as regards the taking of food, exercise, sleep, etc., and the use of such substances as tobacco, tea, coffee, and alcohol. Medicinal treatment is seldom required. Complete rest for a time is sometimes of great benefit in these cases.

The treatment of so-called "soldier's heart" is very unsatisfactory. Absolute rest in bed, and the administration of digitalis and aconite appear to afford the best results. Cardiac asthenia should be treated, according to Da Costa, by rest in bed, followed by graduated shower baths, massage, and Swedish exercises. The diet should be nutritious and liberal as regards quantity. Alcohol is also of service. So far as medicinal treatment is concerned Da Costa strongly advocates the use of strychnine, and, failing this, of arsenic or of the cardiac tonics. Nerve sedatives are occasionally called for. Allbutt suggests that compression of the abdomen by a properly fitted binder or pad might be of use in view of the pathogenesis of this disorder, which he thinks may be a dilatation of the vessels in the splanchnic area.

The treatment of arrhythmia does not require special consideration, inasmuch as it turns on the application of the general principles already indicated. In all cases treatment should be addressed to the cause of the cardiac disturbance, be this digestive disorder, uterine derangement, or the abuse of tea, alcohol, tobacco, coffee, etc. If the cause cannot be discovered, and provided the patient suffers no inconvenience from the abnormal cardiac action, interference by means of drugs usually does more harm than good, and is therefore to be deprecated.

Tachycardia does not offer much scope for treatment, since the conditions on which the attacks depend are not fully understood. The general health of the patient must be promoted by suitable hygienic and dietetic means, and any functional disturbance of other organs which might stand in causal relation to the attacks should so far as possible be remedied. Digitalis exercises little or no influence on the paroxysms, but *strophanthus* has occasionally appeared to do good. The bromides are of service in some instances, and they may be given in combination with full doses of belladonna. The salicylates and iodides are also worthy of trial when other measures fail.

Treatment by electricity has not fulfilled expectations, but the application of the continuous current over the nerve trunks in the neck has sometimes appeared to mitigate the severity of the attacks.

The treatment of bradycardia, when this is necessary, should be directed to the cause of the condition.

In the absence of organic disease of the heart, the feeling of faintness which precedes the occurrence of actual syncope and loss of consciousness can usually be relieved by making the patient sit down and bend forwards with the head between the legs.

When fainting has occurred the patient should be placed flat on his back, with the head at a lower level than the rest of the body. The clothes round the neck, chest, and abdomen should be loosened, and a current of cold air allowed to play on the skin with the object of producing contraction of the superficial vessels and of raising blood pressure.

Reflex stimulation of the heart and respiration is accomplished by flicking the face and chest with a wet towel and by the application of ammonia, smelling-salts, and other pungent substances to the nose. If these measures fail to restore consciousness, ammonia, ether, brandy, or strychnine may be injected subcutaneously, or per rectum.

When the syncopal attack is due to hæmorrhage the patient should be inverted, or the limbs bandaged from the feet upwards, with the object of driving blood towards the heart and brain. Meanwhile, hot applications may be made to the præcordial region.

In urgent cases artificial respiration, galvanization of the phrenic nerves, or even transfusion of a saline solution may be required. After consciousness is restored the patient should remain in the recumbent position until all danger of a recurrence of the attack has passed away.

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